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FOUNDED BY JAMES PLEASANT PARKER

VOLUME 60

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ANNALS
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L

THE TRANSMISSION OF PAIN IMPULSES VIA THE CHORDA TYMPANI NERVE

JAMES B. COSTEN, M.D.
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AND
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The chorda tympani nerve, once described as the sensory part of the seventh cranial nerve, carries preganglionic fibers from the superior salivatory nucleus to the submaxillary ganglion, and thus contributes largely to the secretory activity of the gland. Its terminal part fuses with the lingual nerve and supplies taste sensation to the anterior two-thirds of the tongue. Kuntz[1] reviewed the proof of secretory fibers to the salivary glands by Ludwig in 1851, and by Heidenhain in 1878; they stimulated the chorda by weak induction shocks, causing the submaxillary and sublingual glands in dogs to secrete actively; they were able to vary and regulate the degree of secretory function by stimulation of the sympathetic or the parasympathetic nerves to the glands, alternately. Contemporary investigators confirmed the findings.

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The secretory nerve function of the chorda was therefore based upon stimulation of the nerve and measurement of gland secretion.

Within the period of 1882 to 1902, clinical experiences of Erb,[2] Gowers,[3] Turner, Finney and Thomas,[5] Sherrington[6] and others supported the theory of transmission of taste from the surface of the tongue via the trigeminus. It was based upon complete anesthesia, analgesia and ageusia over the anterior two-thirds of the tongue after intracranial section of the second and third divisions of the fifth nerve for trigeminal neuralgia. Köster[7] reported one of his cases to have recovered the sense of taste equal to the normal side of the tongue, the operated side remaining anesthetic to other sensation. Believing in the fifth nerve route for taste, he explained the recovery as due to establishment of collateral innervation on that side with the ninth nerve.

Cushing,[8] in 1903, reported observations—"completely at variance with Gower's findings"—as well as the then accepted theory of the trigeminal route of transmission. Thorough tests for taste perception were made preoperatively, and at intervals during the recovery period. Areas of intrabuccal anesthesia were mapped after section of the semilunar ganglion, two of which involved complete removal. There was an early and transient disappearance of taste which he attributed to postoperative degeneration and swelling, the removal of the ganglion affecting the fibers of the lingual nerve, and giving a physiological "block" to chorda activities.

He found in some of the cases that temporary loss of taste was accurately limited to the anterior two-thirds of the tongue and was accompanied by anesthesia of the same area. He stated: "Although a large part of this territory of anesthesia should remain absolutely and permanently unchanged, careful examination of the tongue will show that in a certain percentage of cases the anesthesia does not persist in its absoluteness. In several of my cases I have found after some days and in association with return of taste, that certain forms of stimuli, such as the movement of a cotton swab over the surface of the tongue, will give a peculiar tactile sensation which is localizable, anesthesia to pain, temperature and to touch by the hair esthesimeter meanwhile remaining complete.

"It seems not impossible that the chorda, representing as it may the sensory portion of the N. facialis, may contain afferent fibres transmitting qualities other than those of special sense. Köster[7] reports cases of facial paralysis in which the hemilingual hypaesthesia has accompanied the loss of taste on the anterior area of the tongue."

When completed, Cushing's observations ruled out for the first time, the fifth nerve as transmitting taste from either the anterior two-thirds or the posterior one-third of the tongue. Definite proof of the pathway of taste from the anterior two-thirds of the tongue only through the chorda tympani by way of the geniculate ganglion and the nervus intermedius to the pons, came with elaborate observations of Lewis and Dandy[9] in 1930, their conclusions being based on the results of intracranial division of isolated cranial nerves in the patients. Such sections also proved that taste from the posterior one-third of the tongue passed by way of the glossopharyngeal nerve only.

Schwartz and Weddell[10] report preservation of taste in the anterior two-thirds of the tongue in two patients after section of the chorda, and loss of taste in one following cutting of the greater superficial petrosal. They conclude that the latter nerve occasionally transmits taste as an alternate route in place of the chorda. In relation to our present interest, this should mean that a chorda large enough to be seen at operation must carry many sensory fibers other than taste if it does not carry the taste fibers. We have examined one chorda specimen which seems to fit this condition (see below, fiber counts).

Realizing that little information was available on the morphological types and sizes of axons in the chorda tympani, Foley[11] in 1945, reported data on the ratio of sensory to preganglionic visceral motor fibers. The ratio of morphological types of axons was studied in dogs and cats, by sectioning the motor roots and counting the remaining sensory axons. Comparison of the number of sensory nerve fibers remaining in the degenerated nerve with section of a normal chorda, gave the totals of each, myelinated and unmyelinated. He found an average of 1157 sensory axons remains in the chorda of the cat and 2205 in the chorda of the dog. Of these it was found that 18 to 23% were unmyelinated.

Impressions of pain impulses transmitted by way of the chorda were noted by Duchenne,[12] 1850, and Blau,[13] 1879. They stimulated the wall of the tympanum through openings in the drum, with irrigations, cautery, and faradic current, producing taste sensations and prickling of the tongue. Blau reported the observations of Tröltsch who had produced these sensations by pressure on granulations within chronic infected middle ears.

In the case which prompted the following investigation the chorda remained intact, after a radical mastoid operation, lying against the wall of the middle ear cavity and covered only by thin

clean granulations. On mechanical or electrical stimulation of this nerve the patient reported a variety of sensations, including sharp pain, referred to the anterior region of the tongue. In addition to repeated testing of this subject with accurately controlled electrical stimulation, we have investigated the fiber content of a number of human chordas from operations or from autopsy, in a search for histological confirmation of the experimental findings.

REPORT OF A CASE

CASE 1.—J. M., aged 34. The patient has had bilateral otorrhea since infancy. Because of excessive discharge and right-sided headache, radical mastoidectomy was advised, the left ear being less active, and easier to control with local treatment. Radical mastoidectomy was done through a postauricular incision on the right side May 10, 1938, in Barnes Hospital, making a flap from the skin of the external canal. A good result was obtained, with a dry cavity.

He returned at six-month intervals for cleaning of the cavity. At various times when a crust was removed from the middle ear wall, he described a sensation in the right border of the tongue. Close inspection of the middle ear revealed the intact chorda tympani nerve lying across its upper third. Light pressure on the nerve produced a sensation of electric shock in the right anterior two-thirds of the tongue, and firm pressure produced actual pain.

A left radical mastoidectomy was performed on Jan. 8, 1951, and the cavity grafted with skin. A dry cavity resulted after two months.

PHYSIOLOGICAL STIMULATION OF THE EXPOSED CHORDA TYMPANI

The subject's right chorda was available. Repetitive condenser charges or direct current pulses of 1 to 2 msec. duration proved to be suitable stimuli. They were applied by means of a camel's hair brush electrode cut down to a fine tip, wet with salt solution. The patient lay on his left side and under illumination through an otoscope an electrode was applied by hand to three loci along the exposed nerve, as indicated in Figure 1. At position *C* the chorda could be effectively stimulated without activating the seventh nerve trunk, while at position *A* the contraction of facial musculature and the sensation of muscle movement offered a convenient reference value for current thresholds. The nerve was investigated four times over a period of two years, after which an attempt was made to remove the nerve for histological examination.

This attempt failed, due to obscuring of the field by profuse bleeding from the overlying granulations, and the tissue recovered contained no nerve, although at subsequent examination the chorda could not be found. We have however made sections of five human chordas, normal and from mastoid operations, the analyses of which are presented in a later section.

The most pronounced effect of nerve stimulation in the subject studied was, consistently, pain in the anterior two-thirds of the tongue. Touch was almost certainly present, reported as tingling or non-painful sensation of "electric shocks" in the tongue. Taste was not reported from stimulation at position *C*, but was obtained at positions *B* and *A*, with a threshold somewhat higher than that for pain. This taste sensation was rather non-specific, being reported as a metallic taste, similar to that reported during direct current application to the side of the cheek (see below). The failure to obtain taste from the peripheral region of the chorda may have been due to pathological changes following infection and repeated cleaning out of the cavity, which then must have affected taste fibers (presumably the smaller fibers) preferentially.

That our case was not like those reported by Schwartz and Weddell in which taste was transmitted via the greater superficial petrosal nerve rather than via the chorda, is indicated by the elicitation of taste from the proximal region near the trunk of the seventh nerve, where it should have been spared trauma due to infection, and so forth.

On mechanical contact with the nerve, by light touch with the fine-pointed brush, pain in the tongue was uniformly reported. This was transitory, and stopped when, after adjustment precisely over the nerve, movement of the brush ceased. Electric stimuli were then applied at 20 to 60 per second, and in some trials for periods as long as the patient would tolerate, on the supposition that taste might require summation over a period of time in order to be recognized. Such summation was in fact indicated at positions *B* and *A*, repetitive shocks giving the response when one or a few failed to do so. The threshold for taste was then higher than that for pain for short periods of stimulation or even with longer stimulation, and required more summation to be appreciated than did pain. This may account for failure to induce taste with mechanical stimulation and for wide variations in relative thresholds for taste and pain at different trials. These threshold relations indicate that taste fibers are smaller than, at least, the larger pain fibers.* However both sensations, taste and

*In previous work on other nerves, chiefly those supplying afferents to skin and muscle in animals and human subjects without anesthesia, touch has been found to have the lowest threshold, pain a higher threshold to direct electrical nerve stimulation. The ratios of thresholds are 1:4 to 1:7, being lower with longer-duration shocks. The sizes of fibers corresponding to these relative thresholds are 12 to 15 mu for the largest touch fibers, and 5 to 6 mu for the largest pain fibers (14, 15). No information has been found in the literature on the sizes of taste fibers, aside from the inference that they correspond to the majority of fibers in the chorda distribution in man and animals, which shows a peak at 2 to 4 mu.

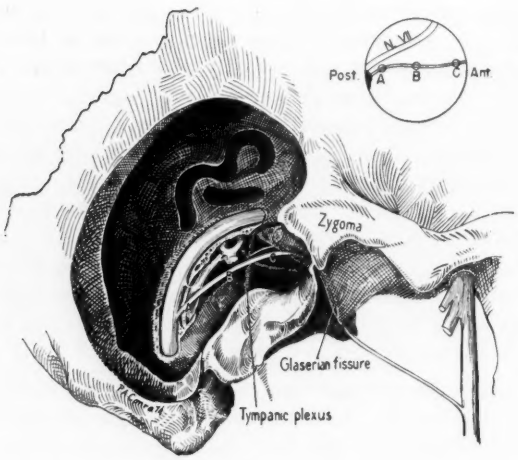

Fig. 1.—Diagrammatic drawing of relations of chorda tympani to middle ear structures, based on dissection pictured in Figure 2. The chorda is shown freed from the tympanic apparatus, as in the subject whose nerve remained intact in this position after mastoid operation. A, B, C, positions at which nerve was stimulated electrically. Inset, diagram showing chorda and N. VII as they appeared on stimulation through the aperture of otoscope placed in external meatus.

pain, were harder to elicit after repeated trials. The thresholds rose, or longer periods of stimulation were required. This might be assigned to central sensory adaptation, but since repeated stimulation resulted in local hyperemia, and the thresholds returned to previous values when the hyperemia disappeared, the effects were probably physical, at the point stimulated.

In terms of threshold for DC current pulses of about 1 msec. duration, and repetitive stimulation, the various sensations showed the following range of thresholds; tingling (touch) and muscle contraction, 0.3 to 0.8 volts; pain in tongue, 0.5 to 3.0 volts; taste, 1 to 6 volts. Variations could be assigned to inaccuracy of contact with the nerve, position on the nerve, and the amount of tissue covering it. With any one placement, and where all three sensations were obtained from the same place, the threshold for pain was three to five times that for touch, and the threshold for taste only slightly higher than that for pain.

Various side effects of stimulation were reported; stimulation over the ampulla caused dizziness, and in the region of the seventh nerve (but probably not from its direct stimulation),coughing was

sometimes induced. Local sensations of touch and pain referred to the ear cavity were reported during placement of the electrode, or on contact with the surface of the cavity, and these local sensations were clearly and consistently differentiated from those referred to the tongue. An auditory "buzzing" which sometimes accompanied repetitive stimulation was presumably secondary to muscle contraction near the ear mechanism, rather than to direct stimulation of the auditory apparatus.

It was also necessary to rule out the possibility of direct stimulation of the tongue by stray currents from the electrodes, since it is known that currents applied to the side of the head can induce a metallic taste in the tongue. In two normal subjects the anode of a direct current circuit was applied immediately behind the ear. A sour or metallic taste resulted at a strength of 7 to 9 milliamperes, if the tongue was in contact with the side of the cheek. With the electrode placed at the angle of the lower jaw the threshold was 2 ma. No pain was reported until considerably higher currents were applied, and this arose from muscles, accompanying strong contraction, and from superficial skin under the electrode. If the tongue was moved from the cheek a much stronger current was required for taste, which then was reported only from the back of the tongue. If the tip of the tongue was then touched to the cheek the taste sensation was reported from the tip. No pain was induced in the tongue.

All these trials indicate that taste under these conditions is due not to direct stimulation of the chorda or the seventh nerve through the skin, but to stray currents flowing through the tongue sense organs, whose electrical thresholds for taste moreover must be very low. But the apparatus employed to stimulate the chorda had 2000 ohms resistance built into it in series with the electrodes, which alone, and disregarding electrode resistance, would limit the current. For instance it would have required 4 volts to give the same spread of current (2 milliamperes) for direct stimulation of taste in the tongue itself, as when the electrode was on the jaw, and 14 volts with the electrode behind the ear. It can be concluded then that the elicitation of pain and taste from stimulation of the chorda at less than one volt was not due to stray currents flowing through the tongue itself.

This test was applied to the subject under investigation and before the second mastoidectomy. On the left side, with a 1.5 mm diameter electrode placed behind the ear, the other on the back, a salty and metallic taste was reported at 2.5 to 3.5 volts DC, starting toward the base of the tongue and reaching the tip at 10 milliamperes of current. With the electrode on the right side, a salty taste was reported with 3 ma. from the middle of the tongue, which spread to

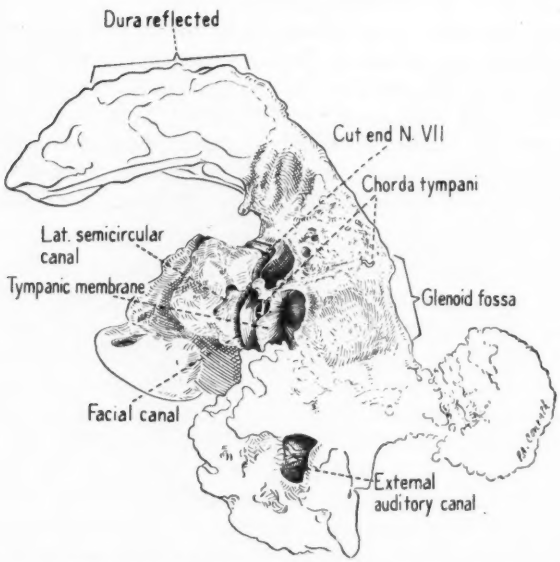

Fig. 2.—Dissection of human middle ear, from autopsy material, redrawn as tracing of photograph to emphasize certain details. The facial and chorda canals were opened and the nerves removed for study. The chorda was cut proximal to its passage across the drum and the portion distal to its exit from the bony canal is in situ. The opened chorda canal is behind and below the free margin of the tympanic membrane. The dissection is from the antero-lateral aspect of the head exposing the middle ear, and involved removal of the anterior attachment of the tympanum with parts of the temporal bone.

the tip along the right side of the tongue at 10 ma. Nystagmus occurred at 7 to 12 ma. from stimulation at either side of the head. No pain or muscle contraction was obtained up to 12 ma. These tests indicate that the subject had reasonably normal taste over the whole tongue, and that direct stimulation of the tongue by stray currents from the vicinity of the ear could not account for the results assigned to chorda stimulation, and particularly for the pain sensations.

Two facts therefore support the conclusion that direct stimulation of the chorda in the ear cavity resulted in the pain and taste sensations reported; when pain was reported from stimulation back of the ear the order of thresholds for pain and taste were reversed from those found on direct stimulation of the chorda; and mechanical stimulation of the chorda gave specific reference of pain to the tongue.

After destruction of the chorda on the right side (the left side having been operated upon, after the physiological tests) the subject

was tested for taste. Normal taste had been present previous to the second operation. It was still normal on the back of the tongue, and absent over the anterior two-thirds on both sides. Temperature sense was still present, and pain to needle prick was present on the posterior third of tongue, absent on the anterior two-thirds; but the patient could differentiate between the sharp lance point, the cut end of an applicator, and a cotton-tipped applicator.

The degree of sensory overlap, and quantitative aspects of the sensory loss including pain and temperature after chorda section are uncertain without a normal side for comparison. Further experiments are planned in which the various sensations will be tested before and after unilateral section of the chorda tympani at mastoid operation.

ANATOMY OF THE CHORDA TYMPANI

In order to obtain further evidence as to the sensory supply to the tongue carried by the chorda tympani, five human and two macaque monkey nerves were fixed in formalin, osmicated and sectioned at 4 to 10 μ by the usual techniques. One chorda and seventh nerve complete were obtained at a fresh autopsy of a man 77 years old who died of cancer in the abdominal cavity. The dissection of this specimen is shown in Figure 2. One normal chorda was obtained at a fenestration operation through the kindness of Dr. Theodore Walsh. Three chordas were removed from infected ears at mastoid operation; these showed no degeneration of fibers or other pathological signs.

A 6 μ section of each nerve was studied in the following manner: One microscopic field was drawn by each of two individuals, in camera lucida, under 4 mm or oil immersion objectives, and at overall magnification of 1000 or 1600 diameters. In each nerve, one of the fields drawn was in a region where, under low power, the greatest number of larger fibers was seen; the other was drawn in the region where the fewest appeared. Most of the nerves showed a considerable difference in different regions, most of the larger fibers being toward one-half of the nerve cross section. The diameters of all fibers on these drawings were measured in millimeters, and counted into groups of 1 mm. width. These measurements were then converted to micra. The results were plotted on co-ordinate paper, with size intervals on the X axis, against numbers of fibers in a group on the Y axis. The numbers of fibers in a field varied from 115 to over 300, depending chiefly on the number of large fibers present. The area of one field, i.e., the area in which fibers could be accurately drawn through the camera lucida aperture, was about ten percent of

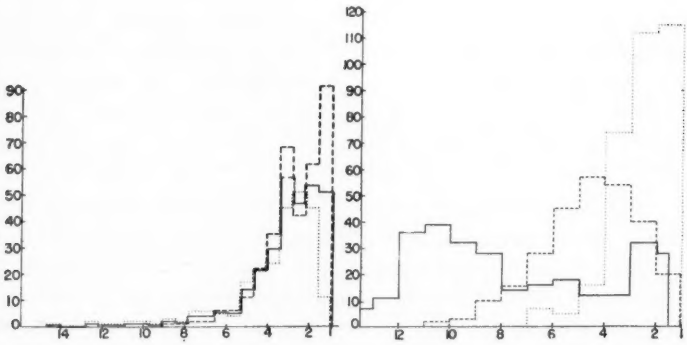

Fig. 3.—(L) Size distribution plots of myelinated fibers of the chorda tympani, normal human nerve from fenestration operation, diameters of size groups on the X axis in μ, numbers of fibers per group on the Y axis. Dotted graph, 251 fibers of one microscopic field in a region of the chorda showing a greater number of larger fibers. Dash graph, 349 fibers of a second field in the region showing the fewer number of larger fibers. Full line graph, the average of these two counts. This fiber distribution is essentially representative of two other chordas similarly counted, in contrast to Figs. 5 and 6. The smaller fiber range of one field, and all of the other field, were counted on X 1600 camera lucida drawings, to the nearest 0.5 mm diameter, and reduced to mu in plotting. The accurary with which fibers can be drawn probably does not justify this close measurement, and most of the nerves were counted to the nearest whole mm. In particular counts below 2 μ are of doubtful accuracy, and might as well have been grouped in one count, 1.5 to 2.5 μ.

Fig. 4.—(R) Dotted graph, chorda tympani of macaque monkey, counts of 329 fibers in one field on a X 1000 drawing measured to the nearest mm. Otherwise as in Fig. 3. Dash graph, counts of 273 fibers in one field of motor area of N. VII, same monkey. Full line, count of 183 fibers in motor area of human nerve VII, from autopsy. The relatively smaller sizes of the motor fibers of the monkey are typical of this animal as compared to cat, dog and man. In addition to afferent fibers, the smaller fibers of both nerves include the group of small motor fibers which innervate the spindles of skeletal muscle generally.

the whole chorda area. No correction was made in either fiber size measurements or in measurements of areas of nerve cross section for shrinkage in preparation, which in our experience with nervous tissue is usually 20 to 30% in one dimension.

On each chart, the counts of the two fields were plotted separately, one in dotted lines and one in dashes. The two counts were then added by size groups, the results divided by two to average, so that the sums could be plotted on the same scale, and the average curves drawn in full lines (Figs. 3 to 6). The five to six hundred

fibers so recorded represent but a small fraction of the totals in the nerves; the consistency of the results may serve as a measure of the adequacy of the counts. Careful inspection of each section under lower power indicated that the differences between the various nerves as shown by the size distributions plotted were real ones. The overall conclusion forced upon us is that the chorda tympani of the human has a variable fiber composition, especially with respect to the larger fibers, those which are of the size which serve touch and pain in other peripheral nerves. In one only of these nerves (Fig. 6) there was a relatively low count in the smaller fiber range, as might be expected if taste fibers took a different route to the tongue, such as via the greater superficial petrosal (Schwartz and Weddell, loc. cit.)

In our normal specimen from autopsy, where the complete seventh nerve was obtained, and in one monkey, serial sections were made of this nerve from proximal to distal through the region of the chorda. At this level the small fibers of the seventh nerve have largely segregated to one margin (Fig. 8), and about one half of this small fiber bundle was contributed to the chorda of the human nerve, and at least two-thirds of this area in the monkey nerve. The cross sectional area of the human chorda was 8% of the total seventh nerve including the chorda above its branching. After exit of the chorda, the area of small fibers remaining was still 8.5% of the total. The main nerve area was 1.47 sq mm, that of the chorda 0.12 sq mm, which is slightly above the average of the areas of the five human chordas sectioned. That of the monkey seventh nerve was only 0.44 sq mm, but the monkey chorda had an area of 0.095 sq mm, nearly as large as the average of the human nerves. The small size of the monkey motor nerve is in part due to the small size of the individual motor fibers in this animal, which are on the average little more than one-half the diameter of the corresponding nerve fibers of the cat, dog and man.[16] In the monkey nerve measured (Fig. 4) the motor fibers showed a peak in distribution of sizes at 4 to 6 μ, while the human motor area counted had a peak at 9 to 12 μ.

Most of the small fiber bundle remaining after the exit of the chorda leaves the main nerve, in both man and monkey specimens examined, in a branch separating just distal to the chorda branch. This presumably comprises the seventh nerve component of the posterior auricular nerve. Complex fiber counts were made of this region in the human seventh nerve before the exit of the chorda and also between chorda and auricular branch, for comparison with the chorda itself (Fig. 7). In both specimens this branch left the

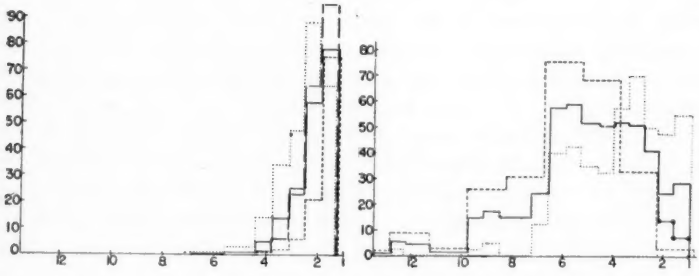

Fig. 5.—(L) Counts of three fields, total of 549 fibers, of chorda tympani removed at mastoid operation. Separate counts, dot, dash and long dash; average of three, full line graph. Otherwise treated as in previous figures.

This nerve showed the fewest fibers in the larger size range, of the five human chordas examined, and is similar to nerves reported by others from cat and dog in this respect. The smaller than average size of this nerve, 0.065 sq mm in cross section, compared to the average for the other four, 0.106 sq mm, may correspond to this relative absence of large fibers, which would occupy more area per fiber in a cross section than smaller fibers. This would imply that the numbers of small fibers in this smaller than usual nerve might be equal to those of the other larger nerves examined.

Fig. 6.—(R) Counts of two fields, 252 and 181 fibers, of a human chorda removed at mastoid operation. This nerve, in contrast to that of Fig. 5, shows a relative paucity of small fibers. See text. Full line graph is average of two field counts as in Fig. 1. The duplication of the full line at right end as marked by open circles is the average of the two counts when one concentrated bundle containing small fibers only is omitted. This bundle, having the histological appearance of an autonomic component, was subtracted from the dotted graph before averaging the two in order to present a plot considered to be more representative of the nerve as a whole.

main nerve just distal to the chorda branch and before the main nerve broke up into motor fascicles. In the monkey it passed through the temporal bone by a separate canal and was traced to a region behind the ear. The fiber distributions in the auricular branches in these two specimens, human and monkey, were not significantly different from the distribution in the chordas, of the same species. It is apropos to point out that the posterior auricular nerve is a sensory skin nerve, and undoubtedly supplies touch and pain fibers to the periphery. Its similarity in constitution to the chorda implies similar modalities carried in the chorda, while its large component of small fibers in the range in which one must infer taste fibers to lie in the chorda raises the question whether many of the small fibers in the chorda may not serve other sensations than taste; for instance,

pain, temperature, etc. Our data are not adequate to more than raise this question. Further, although the posterior auricular nerve is stated to carry sensory fibers to the region of the ear which are contributed to the seventh nerve trunk by a branch of the vagus, no such branch was found in our human autopsy specimen.

Bruesch[17] states that of nine monkey nerves examined in serial sections, three showed significant contributions of the vagus to the seventh trunk; four showed fine twigs which might have come from this source but contributed few fibers; and in two no contribution could be detected. We would infer, from tracing this fine fascicle along the facial trunk to its exit, that most if not all the fibers in the two auricular branches sectioned, one human and one monkey, were of seventh nerve origin like the chorda itself.

The cross sectional areas of our five chordas measured on an average 0.097 sq mm; the seventh nerve cross section above the chorda branch in the one human nerve measured was 1.47 sq mm. One specimen of chorda (Fig. 5) had a cross section of only 0.065 sq mm and conspicuously lacked the usual large fibers in both fields studied and in the nerve as a whole. Comment on this situation is included in the legend. Another nerve (Fig. 6) was of average size, but showed much fewer small fibers than the other nerves. Most of the small fibers in that area of the latter nerve which contained the larger number of them were concentrated in one region of the field. These small fiber fascicles have the appearance of autonomic bundles in peripheral sensory nerves, and may not belong in the sensory component. The counts of this field are at any rate not typical for the whole nerve; and if the small fibers concentrated in one bundle of this field are omitted, the distribution of the remainder is quite similar to that of the other and more typical field counted. This nerve therefore has the appearance and the fiber distribution over most of its area similar to that of such a nerve as the saphenous (Gasser and Erlanger,[18] 1927, who also made counts of the chorda tympani of the dog). We infer that it may have contained less than the usual number of taste fibers, and represents chiefly the general somaesthetic supply to the tongue and adjacent mucous surfaces. It may represent such a case as those reported by Schwartz and Weddell where taste fibers from the anterior region of the tongue were not confined to the chorda tympani.

Such an inference must be based on the identification of some fiber group to which taste can be assigned. In all the nerves studied, and in each of the two fields of each nerve counted, with the exception of the large fiber area of this one exceptional nerve (Fig. 6,

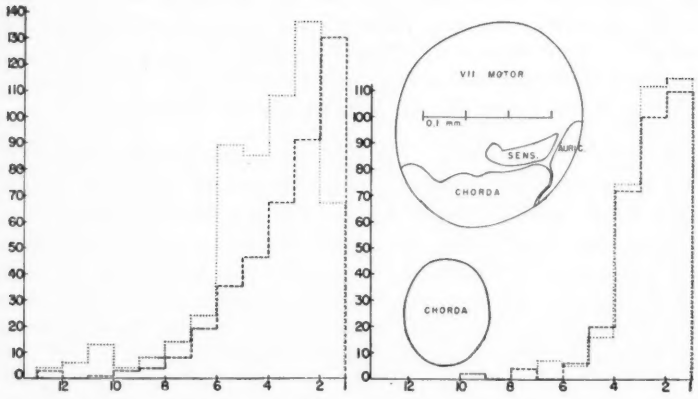

Fig. 7.—(L) Dotted line, plot, of fiber sizes in two microscopic fields, 562 fibers, of chorda area in N. VII trunk from human autopsy. Section is just central to branching of chorda where the fascicles of the chorda could be traced into the trunk in serial sections. Dash line, similar plot, 407 fibers, of the small-fiber area of the nerve trunk distal to the branching of the chorda. Most of this remaining bundle left the trunk in what is taken to be a posterior auricular branch. If there is any significance in the plotted difference between the two graphs, which we believe lies close to the margin of error in size estimations of these small fibers, the auricular sensory nerve has a relatively greater number of smaller fibers.

Fig. 8.—(R) Comparison of one field each of the fiber distribution in the chorda tympani, 329 fibers, and the auricular fascicle of N. VII, 157 fibers, of the monkey nerve. The numbers for the auricular fascicle were multiplied by two in plotting, to bring the graphs to the same height for comparison of form. The low power camera lucida drawings to the left are of the N. VII fascicles, which could be traced in serial sections into the chorda (drawn below), into a branch followed to the region of the ear (auric.), and into other sensory branches (sens.) not followed to their destination.

dash line), a definite maximum occurs in the size distribution in the 2 to 3 or 4 μ fiber range. This is also true of our monkey chorda, and is reported by Bruesch[19] for the cat, by Van Buskirk[20] for the cat and dog and by Foley and DuBois[21] for the cat and dog.

As a further, if rough, means of summarizing the fiber contents of these nerves, the area of a given field could be divided by the number of fibers counted in it, to give the average area of nerve cross section per fiber contained. For instance in the typical chorda large fiber region there is a nerve fiber for each 55 sq μ of the area. For the small fiber region of the same nerve an area of 45 sq μ is assignable to each fiber. For the human seventh nerve motor region

this figure is 120 sq μ. For the one nerve showing a general deficiency of small fibers the figure for one area measured is 55 sq μ, for the other 62, both comparable to the large fiber areas of the other nerves.

DISCUSSION

No attempt has been made by us to estimate the numbers of fibers of different origin, motor, sensory, etc., in the nerves studied, since no degeneration experiments were possible, and such experiments on animals (Foley, Bruesch, Van Buskirk, loc. cit.) have given an adequate picture of the general functional scheme. The findings of these authors that the non-myelinated fibers are few, that the only motor component in the chorda consists of small autonomic fibers, and that these comprise only a small fraction of the small-fiber distribution, removes the necessity, for our purpose, of counts after silver staining, or for concern with motor fibers, in assigning functional significance to the size distributions observed.

The composition of the chorda, and especially the comparison of the fiber size distributions in the different parts of the specific small-fiber bundle passing centrally through the facial trunk, gives a consistent picture. At the division of this component to emerge as chorda and posterior auricular sensory branches the differences between the separate branches are so slight as to suggest that the chorda is one of two or more similar divisions of a facial nerve sensory bundle carrying somaesthetic sense from the surfaces of the mouth, middle ear and scalp; and that through this chorda branch a relatively small group of fibers passes to transmit taste. The sizes of these fibers for taste can be only roughly inferred from the predominance of 2 to 4 μ fibers in the chorda spectrum, which however is supported by such a plot as our Figure 5. This nerve had the fewest large fibers of any obtained, and compares in size distribution to the chordas of cat and dog, as reported by the authors quoted above, rather than to our other human nerves. These authors, particularly Van Buskirk, have made the inference that this small-size distribution must include taste fibers; a point we wish to emphasize is that by no means all of such small fibers need be assigned to taste. In fact Foley[11] reports the ratio of sensory fibers in the chorda to taste buds in the chorda distribution as 3 to 1 in the dog, and 2 to 1 in the cat, and speaks of the profuse branching at terminals near taste buds, which might indicate that even fewer than that proportion served to supply this function.

Further support for assigning taste fibers to this region of the spectrum is supplied by our finding that the electrical threshold for

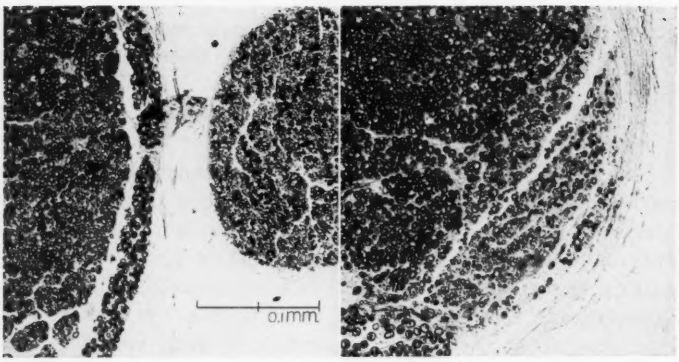

Fig. 9.—(L) Photomicrograph of human N. VII from autopsy showing the recurrent chorda just proximal to its origin from the trunk, and the region in the facial trunk containing the chorda component before division. The superficial crescent of large fibers overlying the chorda area of the facial trunk separates off as a motor branch.

Fig. 10.—(R) Area in N. VII trunk of autopsy specimen distal to the exit of the chorda tympani. Most of this small-fiber area passes into what we infer to be a posterior auricular component, leaving the main nerve just distal to the chorda division but proximal to the branching of the trunk into its motor components. Counts of portions of small fiber areas are presented in Fig. 7.

taste fibers in the chorda is slightly higher than that for pain fibers. This threshold is to be sure that of only the largest fibers of the group serving this function, which will have the lowest threshold. It has been reported previously, on the basis of experiments on human and animal nerve stimulation without anesthesia[14] and from the results of differential block by procaine of human spinal roots[22] that in the peripheral skin and muscle nerve distribution, the larger fibers carry impulses for touch, pressure and muscle sense, and not for pain; that the pain distribution starts only with 5 to 6 μ fibers, and that the largest fibers for temperature sense are slightly smaller than the largest of those for pain in the myelinated fiber range.

Our finding of tactile and pain responses from chorda stimulation corresponds satisfactorily with the findings of such responses at comparable relative thresholds in peripheral nerves. On this basis we infer that in the chorda (and presumably in the auricular complex generally) fibers above 5 μ serve superficial touch, and fibers serving pain lie below this size. This does not mean that no smaller fibers serve touch and pain also, since the method by which thresholds are determined only tests for the largest fibers serving a given

function. The slightly higher threshold for taste than for pain then places the largest fibers for taste somewhat below 5 μ, which is also the range assignable in other peripheral nerves to temperature sense. The evidence for the latter item derives not from relative threshold measurements, but from the order of anesthetic block, for with electrical stimulation a strength which might be expected to activate temperature fibers results in sufficient pain to mask any other sensation. We have no evidence therefore that the chorda carries fibers for temperature sense, but the peripheral distribution of the chorda certainly possesses that sensation. At any rate taste fibers can be said to occupy a similar range in the chorda to that of temperature sense generally.

The chief conclusions we would draw, both from our physiological experiments and from the fiber size counts, is that the chorda tympani of the human, and perhaps of other mammals, is primarily a typical sensory nerve, carrying rather incidentally a component for the special sense of taste. This point we feel needs emphasis because a preoccupation with taste function has characterized most investigations of this nerve. The relatively few sensory fibers distributed to the main motor branches of the facial, and the probable absence of general skin somaesthetic sensory fibers from this distribution, has given the impression that except for the special component of taste fibers the facial nerve was predominantly motor. For instance in Ranson's textbook, discussion of this nerve mentions only two classes of sensory fibers, those serving deep sensibility of the face passing via the motor branches, and those for taste via the chorda tympani. Various authors have mentioned in passing the presumptive presence of other sensory fibers in the chorda, but no specific study of these components has been found in the literature. We conclude that the fibers serving taste are a relatively small fraction of the total.

One of our chorda specimens is quite similar in fiber distribution to the saphenous (and to other skin nerves in general). However, the other chordas studied, and also the auricular branch in man and monkey studied by us, and in fact the whole sensory bundle in the facial trunk, are characterized by a considerably larger proportion of small fibers (4 to 2 μ) than most peripheral sensory nerves contain. This does not necessarily mean that the sensations of touch and pain for instance are proportionally different as compared to the average skin surface, since as noted above the lower limits of the ranges of the various sensations are not known, and it is probable that both touch and pain are carried by smaller fibers than, for instance, the largest fibers serving temperature sense. If we assume

that the various sensory modalities are normally represented in the distributions of the sensory branches of the facial nerve, the large group of fibers at 4 to 2 μ diameter may indicate only that the usual functions are represented in the facial sensory distribution by a greater proportion of smaller fibers. This would correspond to a finding of many more fibers in this range than would be required to supply the number of taste buds reported.

SUMMARY

In a human subject stimulation of the intact chorda tympani has been carried out, after removal of the drum at mastoidectomy. Controlled electrical stimulation elicited touch, pain and taste sensations referred to the anterior two-thirds of the tongue. Final sections of both chordas left this region of the tongue without taste or definite pain, but the subject was still able to distinguish sharp from blunt touch.

Histological sections were obtained of five human chordas, two normal and three at mastoidectomy, and of one monkey, and sample fiber counts were made of each. From the monkey and from one human autopsy, the seventh nerve complex was obtained. Similar counts were made of the sensory bundle in the facial trunk proximal and distal to the division of the chorda. The fiber distribution of the sensory bundle below the chorda branch, mostly passing to a posterior auricular component, is practically the same as that in the chorda tympani.

The typical chorda of our human series (four out of five nerves) contains fibers up to 12 or 14 μ diameter, of the sizes carrying touch impulses in peripheral skin nerves, and fibers in the 6 to 4 μ range which generally carry pain impulses. Comparison of the posterior auricular branches (which are obviously skin nerves) with the chorda tympani suggests strongly that even in the 2 to 4 μ range where taste fibers presumably fall, not all the fibers serve taste. This is the size range in which in other skin nerves temperature sense is represented.

In general the chorda tympani appears to be a nerve of general somaesthetic supply to the surface of the tongue, including the special fibers for taste within the size group usually characteristic of temperature sense.

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LI

INTRACRANIAL DIVISION OF THE EIGHTH NERVE FOR MÉNIÈRE'S DISEASE

A FOLLOW-UP STUDY OF PATIENTS OPERATED ON BY DR. WALTER E. DANDY

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The syndrome of impaired hearing, tinnitus in the affected ear and intermittent attacks of vertigo has been recognized since the days of Aretaeus[1] but was first fully described in 1861 by Ménière[2] and now bears his name.

Various forms of medical and surgical therapy have been used in the treatment of Ménière's disease. Intracranial division of the 8th nerve was first suggested by Charcot[3] in 1894, but Ballance[4] and Frazier,[5] in 1908, made the first clinical report of the end results of this operation. The procedure was popularized by the late Walter E. Dandy[6] who began, in 1924, to section the entire auditory nerve. In 1931 McKenzie[7] sectioned only the vestibular portion of the 8th nerve in order to preserve hearing. This procedure was quickly adopted and repeatedly described by Dandy.[8, 9, 10, 11] The series reviewed in this paper continued up to the time of Dandy's death in 1946 and covered 587 patients with only two deaths. Both fatalities occurred in the immediate post-operative period and were caused by meningitis. The infecting organism was E. coli in one case and pneumococcus in the other. A follow-up study of this group of patients, to determine the late results of this procedure, was made. We were interested especially in finding how many patients who had partial section of the eighth nerve still had useful hearing in the affected ear ten to twenty years after operation. It is well known

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1. Do you have the same attacks you had before your operation?
   Yes ☐ No ☒
2. Are you dizzy? Yes ☐ No ☒
3. How is your hearing? Good ☐ Poor ☐ None ☐
   Right ear ☒ ☐ ☐
   Left ear ☐ ☐ ☒
4. Have you ringing in the ears? Yes ☐ Rt. ear ☐ Lt. ear ☐
   No ☒
5. How much were you improved by the operation?
   100% ☒ 75% ☐ 50% ☐ 25% ☐ None ☐
6. Describe any other complaints since operation.

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7. Will you return for examination? Yes ☒ No ☐

W.S.

Fig. 1.—This is an example of one of the cards used in our mail follow-up group.

that destruction of the membranous labyrinth with alcohol injection, electrocoagulation or the method recently suggested by Lempert[12] will permanently stop vertiginous attacks, provided the diagnosis is correct, but these methods invariably destroy the hearing on the operated side.

METHOD OF STUDY

Letters were sent to each of these 587 patients. All but 13% of these patients had been operated on from five to twenty years previously. It was surprising to us that 150 of these patients came to Baltimore, often from a great distance and at their own expense, in order to have a neurological and otological examination. A questionnaire was sent with each letter which was answered by 258 people who were unable to return for examination (See Fig. 1). The results of both types of follow-up appear in the subsequent tables. It has been a routine procedure since the introduction, in 1931, of the partial section for all patients to have an audiogram before and after operation as well as on later visits. The audiometric studies are of prime importance since most of the cochlear fibers are spared by partial section. This operation was designed to stop the vertigo and preserve or at least not destroy the hearing in the affected ear. We have been fortunate in having all of our tests throughout the period done on the same audiometer, carefully calibrated, in sound conditioned rooms, and most important with adequate masking of the good ear.

The operative technique was repeatedly described by Dandy and need not be repeated. The results of our studies will be presented under their respective subdivisions.

Vertigo is the most disabling of the triad of symptoms in Ménière's disease. It recurs irregularly, often without warning, and may seriously interfere with the work and social activity of the patient. There are many varieties and degrees of dizziness and often the most expert neurologist has difficulty in distinguishing the patients who need a surgical operation from those who will recover on one of the medical treatments. Further emphasis on this point is given by a group of 23 patients with tinnitus and impaired hearing in both ears. To avoid cutting both vestibular nerves, Dandy selected for section of the vestibular nerve the ear in which either the hearing loss or the tinnitus was most pronounced. In our follow-up study only 13 of these patients were free from vertigo, while the symptoms of the remaining 10 were unchanged by operation.

It is clear from the following tabulation (Table I) that it makes no difference whether a complete or partial section of the eighth nerve is performed as far as the relief of vertigo is concerned. Included in the group of partial sections as improved or unchanged are eight patients who continued to have attacks, and whose eighth nerves were cut completely at a later operation by Dandy. All of these patients have been followed up and all have had complete relief from vertigo by the second operation.

Tinnitus is a prominent and annoying symptom of Ménière's disease. Section of the auditory nerve for severe tinnitus has been reported by Krause[13] in 1898, Wallace and Marraige[14] in 1904, Ballance[4] in 1908 and Frazier[15] in 1913. In Table II it is shown that of 114 patients who had section of the entire auditory nerve on the side of their referred tinnitus, only 42 (32.9%) reported complete relief of their tinnitus. This relief is that obtained five to 20 years after operation and not the immediate result, which gives a higher, but possibly misleading, percentage.

Reports from 245 patients who had section of the vestibular fibers only showed a smaller but significant percentage (26.8%) free from tinnitus. The majority of people in both complete and partial sections felt that their tinnitus was unchanged. It is difficult to explain why tinnitus should cease after operation in only 26.8% of those patients with partial section and in 36.9% of those with complete section of the eighth nerve.

Hearing impairment is the third feature of the Ménière's disease triad. All of the patients were tested before and after oper-

TABLE I.—RESULTS UPON VERTIGO:

COMPLETE SECTION

TIME IN YEARS	NO VERTIGO			IMPROVED			UNCHANGED		
	Exam.	Questionnaire	Total	Exam.	Ques'naire	Total	Exam.	Ques'naire	Total
20+	3	1	4	0	0	0	0	0	0
15-19	5	9	14	0	0	0	0	0	0
10-14	12	33	45	0	0	0	0	2	2
5-9	8	32	40	1	1	2	0	4	4
Less than 5	14	3	17	0	1	1	1	1	2
Percent	95.4%	89.6%	91.6%	2.3%	2.3%	2.3%	2.3%	8.1%	6.1%

PARTIAL SECTION

TIME IN YEARS	NO VERTIGO			IMPROVED			UNCHANGED		
	Exam.	Questionnaire	Total	Exam.	Ques'naire	Total	Exam.	Ques'naire	Total
20+	0	0	0	0	0	0	0	0	0
15-19	3	5	8	0	0	0	0	1	1
10-14	29	65	94	1	1	2	0	3	3
5-9	31	71	102	1	4	5	3	3	6
Less than 5	30	17	47	6	1	7	2	1	3
Percent	87.7%	91.8%	90.4%	7.5%	3.5%	5.0%	4.8%	4.7%	4.6%

ation, as well as in the follow-up examination. One point which cannot be overemphasized is that the ear opposite the tested ear must be adequately masked in order to obtain an accurate examination. There is no doubt that the hearing impairment of Ménière's disease is reversible to a certain extent in some patients. For example, Table No. 3 shows the results of a follow-up examination on 64 patients five to seventeen years after operation. In five, the hearing appeared to be better than before operation, but this may not be statistically valid. Twelve patients show no change in hearing five to sixteen years after partial section of the eighth nerve. Only eleven

TABLE II.—RESULTS UPON TINNITUS:

COMPLETE SECTION

TIME IN YEARS	NO TINNITUS			IMPROVED			UNCHANGED			WORSE		
	Exam.	Ques'naire	Total	Exam.	Ques'naire	Total	Exam.	Ques'naire	Total	Exam.	Ques'naire	Total
20+	0	1	1	0	0	0	3	0	3	0	0	0
15-19	0	5	5	0	0	0	5	4	9	0	0	0
10-14	4	12	16	1	0	1	4	24	28	0	0	0
5-9	3	16	19	2	1	3	2	13	15	0	3	3
Less than 5	0	1	1	2	0	2	4	4	8	0	0	0
Percent	41.7%	36.9%	16.7%	16.7%	1.2%	5.3%	60.0%	53.5%	55.2%		3.6%	2.6%

PARTIAL SECTION

TIME IN YEARS	NO TINNITUS			IMPROVED			UNCHANGED			WORSE		
	Exam.	Ques'naire	Total	Exam.	Ques'naire	Total	Exam.	Ques'naire	Total	Exam.	Ques'naire	Total
20+	0	0	0	0	0	0	0	0	0	0	0	0
15-19	1	2	3	0	0	0	2	3	5	0	0	0
10-14	9	17	26	6	6	12	13	35	48	3	0	3
5-9	4	21	25	4	5	9	19	44	63	3	4	7
Less than 5	7	5	12	2	1	3	14	12	26	4	0	4
Percent	23.1%	29.0%	26.8%	13.2%	7.8%	9.8%	52.7%	60.6%	57.7%	11.0%	2.6%	5.7%

patients in the followed group had absent hearing in the operated ear five to fifteen years after vestibular section and 36 patients had worse hearing. This latter group is of special interest now, since present day hearing aids are powerful enough to afford many patients in this group serviceable levels of hearing. This is most gratifying since the aim of all surgery is to accomplish the relief of the main and most disturbing symptoms with as little destruction of tissue as necessary. The results of our findings on hearing are as follows, but no dependence has been placed on patients who were not seen for examination.

TABLE III.

RESULTS UPON HEARING, EXAMINED CASES ONLY.

COMPLETE SECTION

TIME IN YEARS	IMPROVED	ABSENT	UNCHANGED	WORSE
20+	0	1	0	2
15-19	0	4	0	0
10-14	0	8	0	2
5-9	0	7	0	1
Less than 5	0	9	0	0
Total	0	29	0	5
Percent		85.3%		14.7%

PARTIAL SECTION

TIME IN YEARS	IMPROVED	ABSENT	UNCHANGED	WORSE
20+	0	0	0	0
15-19	0	0	1	2
10-14	4	5	6	13
5-9	1	6	5	21
Less than 5	4	2	15	10
Total	9	13	27	46
Percent	9.5%	13.8%	28.2%	48.5%

The loss in hearing following operation proceeded in several ways. When there was a complete, immediate loss we feel that the nerve was probably inadvertently severed in its entirety. In 48 (7.9%) there was a rather sharp high tone loss following operation. This may indicate that these fibers lie closest to the vestibular portion at the point of section or possibly that they are more susceptible to trauma. The chief type of loss is one in which there is no change immediately after the operation, but a slow, gradual decline over a period of years. The majority of the 46 cases whose hearing was worse on subsequent examinations, falls into this group.

The following audiograms are typical of the hearing picture seen in late follow-up examinations (Figs. 2-5).

The most important complication of the operation is facial paralysis. This resulted from trauma and fell into two categories, transient and permanent. The transient ones cleared within a month. The permanent paralyses usually appeared immediately, although a few came on during the first post-operative week. These varied from a mild, partial paresis to a complete peripheral type of paralysis. The following table (Table IV) summarizes for facial palsy.

TABLE IV.

	TOTAL CASES	NO FACIAL PALSY	TRANSIENT	PERM.
Complete section	131	98	21	12
Percent		74.8%	16.0%	9.2%
Partial section	277	256	16	5
Percent		92.5%	5.8%	1.7%

It can readily be seen that there is a higher incidence of facial palsy in the patients who had complete section.

The second complication of interest is the disturbance of equilibrium which results from section of the eighth nerve. All patients were unsteady for a period of time of from a few days to six months. Difficulty beyond that period was extremely rare, except for occasional unsteadiness on rapid turning of the body. Abnormalities can be found in every case when walking in the dark or with the eyes closed. In these situations there is swaying from side to side and usually deviation to the side sectioned. These changes are not clin-

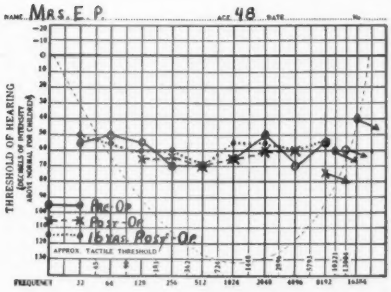

Fig. 2.—This audiogram is rather typical of those patients who showed some improvement in hearing many years after partial section of the eighth nerve.

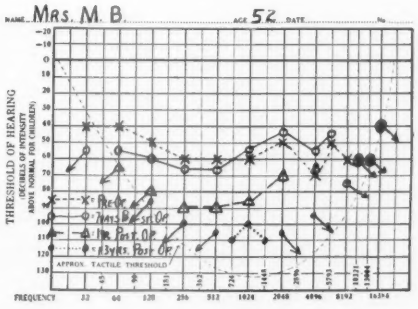

Fig. 3.—This audiogram shows a gradual progressive loss of hearing in the years following operation.

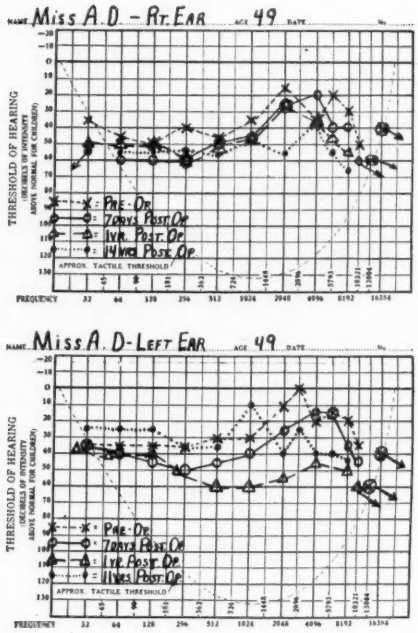

Fig. 4.—One of the bilateral partial sections with both ears shown by audiometric examination several years following each operation.

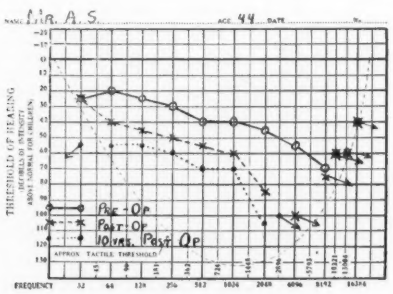

Fig. 5.—An example of high tone loss immediately following operation with a later test for comparison.

ically significant and no patient felt incapacitated by them. The unsteadiness was much increased in patients with bilateral section and usually a year was required for normal walking to return. These people experience great difficulty on walking in the dark unless they are able to use their hands to support themselves.

A characteristic visual disturbance accompanies bilateral sections. Vision is normal when the patient is at rest, but when in motion objects appear to move up and down rapidly. As soon as the patient stops vision returns to normal.

No ataxia on finger-nose or heel-knee test was found in any patient either with unilateral or bilateral eighth nerve section.

We are not making any effort to discuss in detail either the diagnosis or pathology of Ménière's disease. However, it has been thought that in cases where it is difficult to determine from which ear the symptoms arise, the direction of rotation of objects during an attack would provide the clue. In Table No. 5 we list those patients who gave an accurate history pre-operatively.

TABLE V.

	DIRECTION OF OBJECTS DURING ATTACK	
AFFECTED EAR	RIGHT TO LEFT	LEFT TO RIGHT
Right	30	34
Left	33	40

From these figures it can be seen that there is no correlation between the ear involved and the direction of rotation of objects. This agrees with the findings of Crowe.[16]

Several theories of the pathology of Ménière's disease have been advanced. Dandy felt that it involved pressure by anomalous arterial loops compressing the eighth nerve. In this series Dandy noted 85 such loops in the course of his 607 operations.

The other chief theory of the pathology of Ménière's disease, hydrops of the internal ear, was presented by Hallpike and Cairns.[17] The temporal bones have been examined pathologically in only once case in our series, and in this case, changes similar to those described by Hallpike and Cairns were found.

It is also of interest to note that of the 607 affected ears in our series, 365 were in males and 242 in females. The right ear was involved 294 times and the left 313 times.

The age distribution of the cases is shown by the following table:

TABLE VI.

AGE AT ONSET OF SYMPTOMS	NUMBER OF PATIENTS	
0-9	0	
10-19	20	3.4%
20-29	81	13.8%
30-39	168	28.6%
40-49	152	25.9%
50-59	117	19.9%
60-69	48	8.2%
70-79	1	0.2%

One notes from these figures that the disease is chiefly one of middle age.

DISCUSSIONS AND CONCLUSIONS

We do not have a large series of patients treated with a medical regime to compare with this group of surgically treated patients. However, most of these operated patients were previously treated by medical means with failure. We have, therefore, tried to point out only operative results of eighth nerve section and here the relief of vertigo has been excellent. The partial section seems to be favored since it relieves vertigo almost as well as complete section. By this approach, usable hearing has been kept for 40-45 per cent of the ears. Facial palsy is much less frequent when the partial section is used, but tinnitus is not relieved in as many cases.

It must not be assumed that operation of this type is considered the only, or even necessarily the best, form of therapy in this condition. It is our belief that all patients who present themselves with Ménière's disease should have a thorough and complete trial with medical treatment. If the symptoms cannot be relieved by such methods, then eighth nerve section seems to the authors to offer the

best surgical treatment available. This method leaves the most residual hearing of any surgical treatment known. We strongly advise intracranial section of the eighth nerve as the treatment of choice when surgical therapy is indicated in Ménière's disease.

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LII

TEMPORAL LOBE HERNIATION

THROUGH TRAUMATIC DEFECT IN TEGMEN OF TEMPORAL BONE WITH CEREBROSPINAL OTORRHEA

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Pulsation of the posterior canal wall of the ear is an unusual occurrence and led, in the case to be described, to the diagnosis of temporal lobe herniation into the mastoid bone. There are many reports of brain herniation into the temporal bone, but it is a rare complication following mastoid surgery, and even more rare following temporal bone fracture.

Brain hernias due to trauma have been described by Sargent.[1] Roques and Huard[2] report the gross pathology of traumatic hernia in a well illustrated article. An excellent review of the literature can be found in an article by Hall[3] discussing herniation as a postoperative complication in otology. He mentions brain herniations due to mastoid surgery, trepanation for brain abscess, and surgical intervention for lateral sinus thrombophlebitis, as well as those from trauma and those experimentally produced. The most exhaustive studies are by Caboche[4] and Galebski.[5]

Hall states that in regard to the dural opening:

1. Dural opening alone is insufficient to cause a hernia.

2. The size of the opening is important because of strangulation caused by a small opening.

In regard to increase in intracranial pressure:

1. The cause is mechanical in immediate hernias.

2. There is pathogenic duality in secondary hernias. The cause is infection and mechanical action.

He continues that merely opening the dura does not necessarily cause a hernia unless something is present to raise intracranial pressure, cause swelling of brain tissue, or take up intracranial space (abscess, meningitis, etc.).

He points out that hernias through large openings usually do not become strangulated and seldom recede. These usually become covered with skin.

Rand[6] advocates surgery through a clean field into a clean field. He advises against attempting to remove a hernia through the mastoid approach where possible abscess may be found and thus going through a contaminated field into a possibly clean field. The treatment recommended, however, is: the avoidance of trauma, the prevention of infection, and the lowering of intracranial pressure. Removal is condemned because of the possibility of causing meningitis, encephalitis, or hemorrhage, and of opening the lateral ventricle. He states that it was attempted in a few cases but that the herniations recurred and were usually larger than before.

The patient here presented was treated by surgery through a clean field, and in contrast to Hall's experience, the hernia has not recurred during four months' observation. The case was diagnosed and studied by the section of otolaryngology. The patient was operated on by Major George Hayes, Chief of the neurosurgical section at Walter Reed Army Hospital.

REPORT OF A CASE

A nineteen-year-old white male was admitted to Walter Reed Army Hospital on December 16, 1950, with the chief complaint of drainage from the left ear for four days. The history of the present illness revealed that the patient had fallen from a hayloft at the age of ten. He had been unconscious for approximately eighteen hours, after which the left ear was found to be draining. The fluid was purulent when first noted, but became clear as time went on and stopped completely in three to four months. There was no trouble until one week prior to the present admission when he had a mild upper respiratory infection with nasal discharge and congestion. Upon arising the following morning, the left ear was draining clear fluid of a watery consistency. A frontal headache developed on the second day, and the patient was put into the hospital where penicillin was administered. The drainage ceased four days after it had started. There were no other symptoms.

The family history and systemic review were not contributory.

The patient was a well developed and well nourished young white male appearing to be in good health. His temperature was 98.6 degrees and the blood pressure 140/70.

The eye, ear, nose, and throat examination revealed no signicant findings except for the appearance of the left ear. Examination of this ear revealed the posterior and posterior-superior canal wall to be very thin and pulsating regularly with the heart beat. The canal contained white debris, which was not foul. The drum was intact. The middle ear could be inflated by a gentle Valsalva maneuver and,

Fig. 1.—Roentgenogram, normal ear, Mayer's position.

Fig. 2.—Roentgenogram of the present case before operation; Mayer's position.

also, by catheter. Positive pressure with the Brüning otoscope demonstrated that the pulsating canal wall could be ballooned into the mastoid cavity. With the Valsalva maneuver or the catheter inflation the pulsating canal wall bulged even farther into the ear canal. There was no discharge of any nature on admission to this hospital. The annulus tympanicus could be seen and seemed to be completely intact.

The Weber test was referred to the left. The Rinne was positive bilaterally.

AUDIOGRAM

December 18, 1950

Frequency	256	512	1024	2048	4096	5792	8192
Right	5	5	15	5	10	10	10
Left	10	25	55	60	75	75	80

The caloric test was normal on the right side. The left labyrinth failed to respond to the Kobrac test but reacted to stimulation with 30 cubic centimeters of ice water at 38 degrees centigrade in 60 seconds with the head 60 degrees backward. No rotatory nystagmus, past pointing, falling, or vasomotor reaction was produced on irrigation of the left side.

Laboratory studies were all within normal limits. X-rays taken in Waters', Caldwell's, Towne's submentovertical and lateral positions

were not revealing. The Mayer's view revealed a lack of cell walls in the periantral region on the left (Figs. 1 and 2).

The patient was asymptomatic on admission to Walter Reed Hospital. On December 21, 1950, a spinal tap revealed normal spinal fluid under a pressure of 140 millimeters of water. At the suggestion of the neurosurgical department, 5 cubic centimeters of crystal violet dye were placed into the spinal fluid, and, after leaving it there for thirty minutes, a sterile needle was inserted into the bulging area in the posterior canal wall of the left ear. Blue fluid welled into the ear canal and continued to drain for seven days. After four days of otorrhea, the blue color disappeared. On January 3, 1951, left temporal craniotomy was done under endotracheal anesthesia. There were adhesions between the brain and the meninges which were believed to be the result of previous meningitis. A defect 2½ by 1½ cm was found in the tegmen of the temporal bone with brain herniation into the mastoid antrum. The meninges did not seem to follow the brain into the defect. Silver clips were placed on three sides of the defect to mark it on postoperative X-ray films. A piece of crushed temporal muscle was placed in the defect and a piece of temporal fascia and muscle was sutured over it. On January 4, 1951, the patient developed a fever of 102.6°, and a spinal tap revealed cloudy fluid with 2,130 white blood cells per cmm and 67 mg per cent protein. 20,000 units of penicillin was instilled into the spinal canal. Aerobic and anaerobic cultures revealed no growth. During this time the patient received daily medication consisting of 900,000 units of penicillin, 6 gm of sulfadiazine, 1½ gm of streptomycin, and 2 gm of aureomycin. On January 6, 1951, spinal tap revealed only slightly cloudy fluid with 205 cells per cmm, with a differential of 88 per cent polymorphonuclear neutrophils and 12 per cent lymphocytes. The protein was 33 mg per cent. Convalescence from January 6, 1951, was uneventful. The temperature returned to normal and medications were discontinued. Examination of the ear at this time revealed the findings to be as before surgery except that there was no bulging of the posterior canal wall and no pulsation. Catheterization and the Valsalva maneuver were not attempted. The patient was discharged to full military duty on January 30, 1951.

SUMMARY

A case of successfully operated temporal lobe herniation into the mastoid antrum extending into the external auditory canal is presented with a review of the more pertinent literature on the subject.

WALTER REED ARMY HOSPITAL.

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LIII

SPEED OF ADMINISTRATION AS RELATED TO THE TOXICITY OF CERTAIN TOPICAL ANESTHETICS

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This experimental investigation is concerned with the relationship of the rate of administration of certain topical anesthetics to their toxicity and represents a continuation of studies previously reported by one of us (HJR)[1] on the pharmacologic properties of topically applied cocaine and tetracaine (pontocaine^{R}).

Hatcher and Eggleston[2] emphasized the importance of speed of administration by demonstrating that if cocaine is injected slowly intravenously, animals will survive considerably greater amounts than if it is administered rapidly.

Schumacher[3] explained this by showing that death occurs when a certain concentration of drug is reached in the blood stream. This blood level is approximately the same regardless of the route of administration. A rapid intravenous injection results in attaining the critical level quickly. It is reached more slowly and requires more drug with slower administration. Many times the rapid minimum lethal intravenous dose may be given subcutaneously because slow absorption delays attaining of lethal concentrations in the blood stream.

Actual rate of administration is not the only factor in determining the amount of drug which may be safely given. Speed of detoxication is likewise important. Eggleston and Hatcher[4] demonstrated that this varies greatly with different local anesthetics. Procaine, for example, is destroyed quite rapidly in the cat, a just sublethal dose being detoxicated in twenty minutes. Cocaine, on the other hand, is metabolized slowly, at the rate of about one near fatal dose an hour. This means that slow intravenous injection in

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the cat permits the administration of almost three minimum lethal doses of procaine in one hour but only one of cocaine. Fussganger and Schaumann[5] found that tetracaine lies between these two drugs in rate of excretion.

In this study cocaine and tetracaine were fractionally instilled intratracheally in rabbits over different periods of time and minimum lethal doses noted. The intratracheal route was selected because it simulates the preparation of patients for bronchoscopy.

EXPERIMENTAL PROCEDURE

Rabbits weighing seven to eight pounds were used. The rabbit is completely immobilized in a standard box, the mouth held open by a perforated tongue blade, and a shortened number eight catheter is threaded through the larynx into the upper trachea. Presence of the catheter in the trachea is established by expelling air bubbles in a cup of water during expiration. Once the catheter is in position the animal offers little or no resistance, and solutions may be instilled at the desired rate: Anesthetics tested were cocaine 10% and tetracaine 2%.

FINDINGS

SPEED OF ADMINISTRATION

	COCAINE		
	15 Seconds	2 Minutes	10 Minutes
Minimum	22-24	27	27
Lethal dose		TETRACAINE	
In Mg/Kilo	3.2	4.4	4.4

DISCUSSION

Intratracheal injection of cocaine and tetracaine in fifteen seconds is tantamount to emptying the syringe and catheter as rapidly as possible. More anesthetic is required to cause death with instillation in two minutes because slower administration keeps blood concentration at a lower level. Most significant is the observation that minimum lethal doses are the same whether instillation is made in two minutes or ten. Both of these intervals are considerably longer than circulation time, which precludes the possibility of an acute rise in blood concentration. It may be assumed that similarity in minimum lethal doses is due to no recognizably greater detoxication in ten minutes as compared to two.

As previously noted, detoxication of cocaine in the experimental animal requires at least one hour, tetracaine somewhat less. Virtually the same amounts of unmetabolized anesthetic are hence in the tissues at the end of two minutes or ten. That man, whose tolerance for these drugs is notably lower than that of experimental animals can detoxicate cocaine and tetracaine even as rapidly is questionable.

These observations are not to be construed as suggesting that caution be abandoned and that, in clinical application, patients in preparation for peroral endoscopy be anesthetized in two or three minutes instead of the usual ten or fifteen. The chief value of slow administration, however, does not lie in being able to administer more drug safely but rather in protection of hypersensitive individuals. Patients with hyperergic responses to cocaine or tetracaine tolerate less drug than that customarily given, and rapid administration of a presumably safe dose may quickly overwhelm them. Slow fractional instillation allows time for a possible reaction to occur before the entire amount has been irretrievably given, with infinitely greater assurance that recovery will ensue than if the total amount is given quickly. The administration of topical anesthetics slowly with the thought that a greater quantity may thereby be tolerated affords a false sense of security. Detoxication of cocaine and tetracaine over a practical anesthetizing period such as ten minutes is too slow to affect significantly the total dose which may be safely administered.

In these studies and in all tests involving mucus membrane,[1] reactions from overdosage invariably appeared much more rapidly with tetracaine than with cocaine. This is apparently due to the vasoconstricting action of cocaine, which delays its absorption. The minimum lethal dose of cocaine is of course just as lethal as that of tetracaine, but it requires several minutes longer for a reaction to occur. Although the more quickly appearing reaction of tetracaine might seem objectionable, it actually affords the decided advantage of enabling the operator to withhold further anesthetic in the event of reaction, whereas in the case of cocaine, additional drug might easily be given before a developing reaction is clinically evident.

SUMMARY AND CONCLUSIONS

1. Cocaine and tetracaine were instilled intratracheally in rabbits fractionally at different speeds.

2. Minimum lethal doses were the same whether administration was made over a period of two minutes or ten.

3. Detoxication of these drugs occurs so slowly that fractional instillation over a practical anesthetizing period such as ten minutes

does not safely permit the use of a greater quantity than would be administered in a shorter period of time.

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LIV

EROSION OF THE ESOPHAGUS BY AN INTRATHORACIC GOITER

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Symptoms of dysphagia and dyspnea due to intrathoracic goiters have been reported at frequent intervals in the medical literature.[1-3] Wuhrmann reported dysphagia in approximately four percent of ninety one cases of intrathoracic goiters.

Erosion of the trachea by large intrathoracic goiters, with dyspnea, has been reported by Moersch.[4]

Grief,[5] Jorge,[6] and Baldenweck[7] have cited instances of invasion of the trachea and larynx by adenomata of the thyroid.

A survey of the medical literature fails to reveal any instance in which erosion and penetration of the esophagus has occurred due to an intrathoracic goiter.

Lahey[8] feels that intrathoracic goiters originate as adenoma that usually arise in the isthmus or either lower pole of the thyroid gland and eventually descend along the course of least resistance between the fascial planes into the mediastinum. He feels that intrathoracic goiters rarely produce any pressure changes on the esophagus.

Porter[9] reported a case of intrathoracic goiter which surrounded the trachea and almost completely surrounded the esophagus. Removal of this mass at autopsy demonstrated no esophageal erosion.

Pemberton[2] reported a huge substernal and retrotracheal goiter with marked displacement of the esophagus and trachea without erosion of the esophagus.

Berard and Mallet-Guy[3] describe intrathoracic goiters in two cases which were so adherent to the esophagus that the esophageal

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Fig. 1.—A-P view of the neck and chest showing a large substernal mass. Note the areas of calcification.

wall was torn during surgery, with resultant post-operative fistulae. Esophagoscopy was not performed in either case prior to surgery.

Santy and Berard[10] removed a mediastinal goiter weighing 300 grams that produced radiologic evidence of esophageal compression that was verified at operation. Here, again, there was no evidence of invasion of the esophageal wall by the mass.

REPORT OF A CASE

A 64 year old white male laborer was admitted to the Otolaryngology Service of the St. Louis City Hospital on February 7, 1951, with complaints of dysphagia and discomfort in the throat of 24 hours' duration. His difficulties followed the ingestion of a bolus of meat. The patient also stated that he had had a goiter for 25 years.

Physical examination revealed puddling of mucus in the pyriform sinuses and a large, firm mass in the neck which was apparently a greatly hypertrophied thyroid gland.

X-ray examination of the neck failed to reveal the presence of a foreign body in the esophagus.

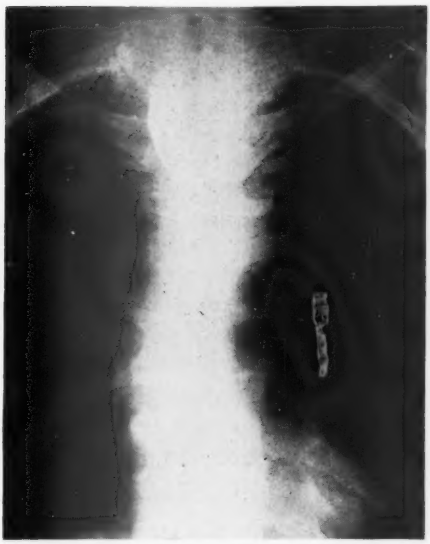

Fig. 2.—Barium swallow showing irregularity of esophageal mucosa below the cricopharyngeus, and marked deviation of esophagus to the right.

On February 7, 1951, an esophagoscopy was performed. The adult Jesberg esophagoscope was employed and the cricopharyngeus was passed with ease. A moderate sized bolus of meat was discovered and removed in one piece through the esophagoscope. Four cm below the cricopharyngeus on the left posterior esophageal wall a smooth mass was noted protruding into the lumen of the esophagus. The area around the mass was clean and no blood was noted. A portion of the mass was removed for study. The bleeding that followed was controlled with adrenalin sponges.

Pathological report: "The section reveals a fragment of necrotic tissue debris with loss of all identifiable architecture and large fragments of homogeneous pink-staining material having the appearance of colloid fragments of thyroid tissue with small calcific foci. No esophageal wall or mucosal lining was seen. Diagnosis: Tissue from esophagus, nodular involutional thyroid with fibrosis, and a bit of foreign body material."

Following the esophagoscopy, the patient developed a fever of 100.4°. A small area in the anterior part of the neck became crepitant; however, no dyspnea was observed and repeated examination failed to reveal evidence of mediastinal emphysema. Following the

use of penicillin, streptomycin, and terramycin, the fever and subcutaneous emphysema disappeared in 48 hours.

On February 20, 1951, a barium swallow examination of the esophagus was performed.

X-ray report: "The trachea and esophagus are deviated to the right and posteriorly by a large superior mediastinal mass which is partially calcified and bulges into the anterior cervical region and over the manubrium of the sternum. Irregularity was noted in the mucosa of the esophagus on its posterior left medial aspect. The enlarged superior mediastinal mass, projecting in the cervical region with displacement of the esophagus, is thought to be due to an enlarged substernal thyroid gland and the irregularity of the esophageal mucosa would indicate esophageal erosion."

The Surgical Service recommended surgical removal of the intrathoracic goiter, but this was refused by the patient. He was discharged, symptom-free, on February 23, 1951.

SUMMARY

1. A case of erosion of the esophagus by an intrathoracic goiter is presented.

2. A review of the literature fails to reveal a previously reported case of invasion of the esophagus by an intrathoracic goiter as proven by esophagoscopy and biopsy.

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LV

INFERIOR MEATAL ACCESSORY OSTIA

REPORT OF A CASE

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Variations in the typical anatomical relationships of the paranasal sinuses are frequently found. The frontal sinuses are usually situated in the vertical portion of the frontal bone, but may extend into the orbital portions or may be so tiny that x-ray visualization is difficult if not impossible. The ethmoids are so unpredictable that no standard location of the anterior or posterior cells can be postulated, and extensions into the middle turbinate, parasphenoid region, and the agger nasi region are not unusual.

The sphenoid sinuses also vary widely in size, shape, and position, but not to such an extent as the ethmoid cells.

The maxillary antrum probably varies less in its anatomical relationships than any other of the sinuses. In the adult the shape is usually pyramidal, and the ostium is usually situated high on the medial wall, opening into the middle meatus of the nose, more specifically into the hiatus semilunaris. Accessory ostia are not uncommon, occurring in 25 to 30% of patients, but they invariably open into the middle meatus. No record of the natural opening into the inferior meatus has been published to the best of our knowledge.

The ostia occur where the lateral bony wall of the nose is deficient and therefore where the sinus mucous membrane and that of the nose are in apposition. The location is such that protection from inspiratory air currents is afforded. The ostia vary in size and shape, but in general they are from 1 to 22 mm. in length and 1 to 6 mm. in width. They may in occasional cases entirely replace the lateral wall of the middle meatus. They are usually oval or elliptical in shape.

The development embryologically is interesting and essential for a full understanding of the anatomy and the physiology. There

are two theories advanced. Proetz believes that the two opposite bony surfaces are forced to separate from each other in the development of the face and skull leaving a space which becomes the sinus. Following establishment of this space, the sinus is 'sucked in' from the nearest air cavity. The other theory is based on the invagination of the mucosa of the nasal cavity or the so called pneumatizing drive of the mucosa.

In any event the maxillary sinus begins as a pouch of mucous membrane in the floor of the infundibulum ethmoidale on approximately the seventieth day of fetal life. The sinus enlarges with the development of the facial bones and the tooth buds. At birth the antrum is at the level of the upper border of the inferior meatus, and during the course of the next few years assumes a close relationship with the inferior meatal wall. At the age of 15 years the antral floor is lower than the nasal floor.

REPORT OF A CASE

This patient, a 24 year old white female, was first seen when she reported for a physical examination prior to being accepted as a flight nurse. She had no specific complaints relative to her nose except for slight nasal stuffiness on the left side during colds. This had been noted during the last sixteen months. Intranasal examination revealed the presence of a small pale polyp in the inferior meatus on the left. There were no other significant findings. The polyp was removed and a large opening approximately 6 by 8 mm in size was noted in the inferior meatus. The latter communicated with the antrum. A similar but smaller opening was then noted on the right. Both these openings had all the characteristics of a normal ostium. A nasopharyngoscope was inserted and revealed the sinus and its membrane to be normal in all other respects. The middle meatal regions were explored with an antrum cannula and the normal ostium was easily entered. Argyrol was instilled into the sinus by way of the normal ostium and appeared immediately in the inferior meatus. X-ray examination of the paranasal sinuses revealed no evidence of disease in any of the sinuses.

The patient had had a nasal polyp removed from the left side in 1943 and subsequently was treated for sinusitis at weekly intervals for five months. At no time, except for the polypectomy, was any procedure done which required either general or local anesthesia. No antral punctures were ever done. She had no nasal treatments prior to this and none subsequently. She has had two to three colds each year. There was no suggestive allergic history obtained.

In summary, a case is reported in which accessory ostia of the maxillary sinuses were present in the inferior meatuses. Such an anomaly is difficult to explain embryologically, but in the absence of any evidence indicating an acquired condition, their presence must be assumed to be on a congenital basis.

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LVI

THE TREAMENT OF ACUTE SUPPURATIVE OTITIS MEDIA

THE RELATIVE MERITS OF CHEMOTHERAPY AND MYRINGOTOMY IN AVOIDING SURGICAL MASTOIDITIS

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Since the advent of the sulfonamide drugs and the antibiotics, much has been written regarding their role in the treatment of acute suppurative otitis media and their prevention of surgical mastoiditis. There is no doubt that acute middle ear infections have responded remarkably well to these drugs, but whether or not these agents should be used routinely in the treatment of acute suppurative otitis media is controversial.

PRELIMINARY REFERENCES

Horan and French[1] in 1938, reported a marked decrease in incidence of mastoidectomy due to the use of chemotherapy. Before the introduction of sulfonamides 138 of 607 patients (22.7 per cent) developed acute mastoiditis, while in a later group of 155 patients, treated with sulfonamides, only 7 or 4.5 per cent developed acute mastoiditis. House[2] in 1946, reported the incidence of mastoidectomy in two series of cases of acute otitis media. Of 1,241 patients not treated with sulfonamides, 45.9 per cent or nearly one-half of these cases required mastoidectomy, while in another group of 193 patients following the use of sulfonamide compounds 17.6 per cent required simple mastoidectomy. He concluded that the incidence of complications in acute otitis media had been reduced by 20.0 per cent, and that the incidence of mastoidectomy in patients hospitalized because of acute otitis media had been reduced by two-thirds. On the other hand, Page[3] in 1934, reported that prior to the introduction of chemotherapy only 6.0 per cent of 300 cases of acute infection of the middle ear in which myringotomy was indicated required mastoidectomy. Richardson[4] in 1942, reviewed 625 cases of acute otitis media which he followed from paracentesis

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to complete healing in an attempt to evaluate the treatment of acute otitis media with or without chemotherapy. The incidence of mastoidectomy was only 5.3 per cent in the entire series where chemotherapy was not used. Butler[5] in 1948, studied the results of 461 cases of purulent otitis media seen in an army clinic and treated without any chemotherapy. Of this entire group only two mastoidectomies were done.

Many writers have condemned the use of chemotherapy in acute middle ear disease but have stressed its therapeutic value in the treatment of complications of mastoiditis, such as lateral sinus thrombosis, meningitis, labyrinthitis or brain abscess. Maybaum, Snyder and Coleman[6] in 1939, not only condemned the use of chemotherapy in suppurative otitis media, but further believed that its use is contraindicated during the course of suspected mastoiditis before and after operation, unless an otitic complication such as meningitis, sinus thrombosis or brain abscess had been diagnosed. Lindsay[7] in 1941, reported that the experience at the University of Chicago Clinic during the past three years had been that the number of operations necessary for acute mastoid suppuration had dropped materially, but the incidence of complications of acute suppuration of the middle ear and mastoid did not appear to have fallen in the same ratio. The total number of complications treated in the season of 1939 and 1940 in this clinic was greater than in any preceding year.

Babcock[8] in August, 1940, expressed the opinion that the effect of sulfonamides in acute otitis media is overrated. Fowler[9] in 1941, stated that acute suppurative otitis media is usually self limited and 80.0 per cent of unselected clinical patients recover in less than four weeks, and if the ear canal is kept clean, the nose shrunken and the eustachian tubes properly cared for, even a higher percentage of these patients recover in this time. He strongly objected to the indiscriminate use of sulfonamide drugs to patients with otitis media and stated that such treatment "reflects ignorance, laziness or poor judgment on the part of the physician." Chemotherapy usually masked the classic symptoms of progressive otitis media and operation was the only remedy for any undrained necrotic focus.

Horan and French[1] concluded that their results in the use of chemotherapy in acute otitis media justified its use, for even if mastoiditis developed, no harm had been done and the patient's resistance was in no way impaired. However, Lyons[10] has shown that delayed responses and a tendency to recurrences of the infection predominate in those patients who have no antibacterial antibodies at the time of treatment and who fail to develop antibodies subse-

quently. Kolmer[11] stated that there is an important relationship between chemotherapy and immunity insofar, at least, as the bacterial infections are concerned. Converse[12] has shown that animals fail to acquire protective antibodies to streptococcic infections if treated with sulfonamides. Gilmore[13] believes that the patient does not acquire a natural immunity when the drugs are used, and the organisms regain their former virulence when the concentration of the drug is lowered.

There are also other factors which complicate indiscriminate use of chemotherapy and antibiotic drugs such as: alteration of the clinical picture of mastoiditis, development of resistant organisms, interference with development of immunity and toxic and allergic reactions.

It is a known fact that the type and virulence of the predominating organisms and their predilection for involvement of the middle ear cavity vary greatly in epidemics of acute upper respiratory infections. Tobey[14] stated that not only do such epidemics vary in virulence but they may show a distinct cycle beginning with a mild otitis and gradually increasing to a peak of virulence and then subsiding. Fowler[9] believes the virulence of the organisms causes the incidence of otitis media to vary from year to year. Henry and Kuhn[15] reported their results of bacteriologic study in 468 cases of acute middle ear infections in patients ranging in age from three months to 63 years. Three hundred sixty-five patients were seen before spontaneous rupture and had a myringotomy, while 103 patients when first seen had draining ears as a result of spontaneous rupture. They stated that many physicians use chemotherapeutic drugs empirically in certain types of infection, and in this series of cases, if these drugs had been given without determining the type of organisms, 47.0 per cent of the patients would have received little or no benefit. Page,[3] Richardson[4] and others have shown that the incidence of mastoidectomies are greatest in hemolytic streptococcal and pneumococcus Type III middle ears.

There is no doubt that the use of these drugs in acute upper respiratory infections has reduced the incidence of middle ear involvement, thus reducing the frequency of surgical mastoiditis. However, it is apparent from the foregoing remarks that the incidence of surgical mastoiditis has not been appreciably reduced by chemotherapy when acute suppurative otitis media is present. Thus the reduction in the number of surgical mastoids is only relative.

The hope has been expressed and it is the belief of many physicians that these drugs will eventually make acute surgical mastoid-

itis practically disappear as a clinical entity, thus myringotomy is frequently delayed or even advised against usually by the general practitioner and the pediatrician because sole reliance is placed on the use of these agents. As long as acute colds, sore throats and contagious diseases continue to plague the human race, suppurative otitis media will be a prevalent complication. So called "primary mastoiditis" without involvement of the tympanic cavity is extremely rare, and if sole reliance is placed on chemotherapy to cure the middle ear infection without obtaining adequate and early surgical drainage by myringotomy, then acute mastoiditis and its serious complications requiring surgery will be relatively as frequent, if not more so, than prior to the advent of these therapeutic agents. Bilchick and O'Kane[16] recommended routine administration of sulfonamides under careful supervision in all cases of otitis media, but in addition, incision of the tympanic membrane should be carried out when indicated, irrespective of administration of the drug, for too much reliance on the drug alone will result in frequent disaster. Richardson[4] reported only a 5.3 per cent incidence of mastoidectomies in a large series of cases of acute suppurative otitis media where paracentesis was performed and no chemotherapy used. Williams[17] in 1933, reported that 10.8 per cent of 14,733 scarlet fever patients from 1922 to 1929 developed otitis media, and of these 10.8 per cent required mastoidectomy. The incidence of surgical mastoiditis was only 6.6 per cent where the drum membrane was incised and no chemotherapy given, as compared to 9.3 per cent where spontaneous rupture occurred. Again in 1935,[18] Williams reported on 427 cases of otitis media in measles. There were 651 affected ears with a total of 41 patients or 48 mastoidectomies. Three hundred twenty-one ears that were incised required 13 or 4.4 per cent of mastoidectomies while 294 ears without paracentesis, 33 or 11.3 per cent required mastoidectomies and concluded that early myringotomy in acute suppurative otitis media in measles had a distinct tendency to prevent development of surgical mastoiditis.

Hall[19] concluded that mastoiditis is much less frequent after paracentesis. Of 221 patients who had myringotomy only 28 required mastoidectomy, while of 189 surgical mastoids only 28 had paracentesis. He also stated that paracentesis not only seemed to shorten the duration of the disease, but had a definite influence on reducing mortality for most fatal cases did not have a paracentesis. Salinger[20] reported that penicillin in acute otitis media was considered of value if administered intelligently and controlled, but stressed the necessity of evacuation of the pus. Bozer[21] in his study of 105 consecutive mastoidectomies which occurred at the Buffalo Chil-

dren's Hospital during 1945 and 1946 concluded that myringotomy should be performed early when there is fluid in the middle ear, and chemotherapy and antibiotics should not be relied upon to cure bone infection in order to avoid operation.

PRESENT INVESTIGATION

The present report is prompted by recent experience on my service at the Massachusetts Eye and Ear Infirmary. During the months of April, May, June and July, 1948, approximately 45 cases of acute suppurative otitis media were seen in the ear out-patient department and were treated by early myringotomy with and without chemotherapy and all recovered without a single mastoidectomy. During this same period, there were admitted to the house service, 30 patients with acute mastoiditis requiring a total of 36 simple mastoidectomies to eradicate the disease. The clinical course, the clinical status of the patients on arrival at the hospital, and the type of treatment utilized will be described in detail.

The patients ranged in age from five months to 16 years. Of this series, ten or 33.3 per cent were under one year of age; 7 or 17.0 per cent from one to five years; two patients or 0.66 per cent between five and ten years; and the remainder ranged from ten to 16 years of age. It will be noted that approximately two-thirds of all cases were ten years of age or under. This point is very important to bear in mind, for when the most common symptoms of middle ear infection such as pain, fever or discharge subside, there frequently remains residual pathology associated with tinnitus, fullness in the ear or even impaired hearing. An adult will soon seek further medical attention for any of these complaints, and by so doing will often avoid mastoiditis, but a child or an infant rarely, if ever, complains of such symptoms. Even the most observant parent may fail to notice the change in the child's hearing unless the process is bilateral causing marked deafness; thus the infected process has ample time to extend to the mastoid cavity before further medical advice is sought.

Almost all patients gave a history of a head cold prior to the onset of the first ear symptoms. Three of these occurred during measles and one immediately after scarlet fever complicated by pneumonia.

All cases presented a history of at least one attack of spontaneous discharge either unilateral or bilateral, and several reported many such recurrences before their first appearance at the hospital for examination.

The original onset of either pain or discharge from the ear prior to the first hospital admission varied from two days to 21 months.

This is illustrated by the following case: R. C. Unit No. 572952, a two year old child with acute bilateral discharging ears was treated with mercurochrome ear drops and saline irrigations for four months. During this time an adenoidectomy was performed without improving the discharging ears. Then one-fourth of a sulfanilamide tablet was given every four hours for four days and the ears became dry. However, five days later both ears again discharged spontaneously, and two months later the patient was readmitted to another hospital where a second adenoidectomy was performed, and approximately 2,000,000 units of penicillin was administered. Again the discharge ceased and the ears remained dry for four days only to be followed by a recurrence of the spontaneous drainage from both ears. This persisted until the child was admitted to the Massachusetts Eye and Ear Infirmary, 21 months after the onset of the middle ear infection. A bilateral simple mastoidectomy was performed, supplemented with large doses of penicillin. The mastoid cell partitions, especially on the right, were soft and the antra and middle ears were filled with infected granulations. Ten days later the patient was discharged home with ears dry and post aural wounds healed.

With two exceptions all patients were treated with insufficient doses of widely different combinations of chemotherapy. Most of them were treated with oral penicillin for only one to several days; others by an injection or two; and a few by penicillin ear and nose drops. Still others had sulfonamides either in tablet or liquid form, while one child was treated with sulfathiazole chewing gum for two weeks. All had received insignificant therapeutic amounts of sulfonamides or penicillin or both, early in the disease, and a few received large doses after the mastoiditis had developed. Yet there was no satisfactory response to this type of chemotherapy because the offending organisms were insensitive to the drug and there was a lack of sufficient drainage. Two brief case histories with serious complications are presented to emphasize these points.

CASE 1.—About Christmas of 1947, W. M. Unit No. 614258, a five year old boy developed a head cold soon followed by pain in the left ear which persisted for two weeks and then subsided, without any drainage. About seven weeks later the child developed scarlet fever with a recurrence of severe pain in the same ear for two days. The patient was treated with penicillin troches for four days. One week later pneumonia complicated the picture, and the left ear drum ruptured spontaneously, releasing profuse purulent discharge. The ear continued to drain until June 2, 1948 when the patient was brought to the hospital because of a marked left post aural swelling, and a flaring auricle of 12 hours duration. A left simple mastoidectomy was performed and pus under pressure released from

the antrum and the zygomatic cells. All cell partitions were soft and the cells contained granulations. The sinus plate was eroded and the sigmoid sinus was also covered with granulations. Virulent beta hemolytic streptococci were obtained by culture. The patient was found to have a moderately severe acute glomerulonephritis and was therefore transferred to the pediatric service for appropriate medical care. He gradually improved and 17 days later was discharged home on bed rest.

CASE 2.—S. W. Unit No. 618642, a five months old female infant developed an acute upper respiratory infection, and was treated with three-fourths of a teaspoon of sulfadiazine every four hours for three days. Two weeks later she was brought to the hospital by the mother because the child had "a crooked face and a swelling of the ear." Local examination revealed a marked left post aural swelling with displacement of the auricle, left facial paralysis and an ear canal filled with purulent discharge of only two days duration coming from a nipple perforation in the tympanic membrane. The x-ray showed a rather large, well developed mastoid with complete destruction of the cells. A simple mastoidectomy was performed and revealed pus coming from a large perforation in the mastoid cortex. The cell partitions were very soft throughout, the cells were filled with pus and granulations and extended well posteriorly and retrofacially. Large doses of penicillin were given post-operatively and the child made an uneventful recovery and was discharged well in ten days.

Not a single patient in this series of mastoid cases had had the benefit of proper drainage by an early myringotomy. During these same four months approximately 45 children appeared in our ear out-patient clinic early in the course of acute middle ear disease and all obtained drainage by myringotomy. Only a few of these children were given chemotherapy. They were all discharged cured within a relatively short time. Not a single patient of this group had to be admitted to the hospital for mastoidectomy.

Most patients were brought to the hospital by their parents because of frequent recurrent attacks of spontaneous discharge from the ears. Few complained of the usual signs and symptoms of acute mastoiditis. Pain and fever were conspicuous by their absence. Several were brought in with post aural swelling. Almost all cases presented thick purulent discharge with nipple perforations, and one third had definite sagging of the posterior superior canal wall. Patients with dry ears had thick, dull, gray drums with obscure landmarks. The x-ray of the mastoids ranged from marked increased density to complete cellular destruction.

LABORATORY DATA

On admission to the hospital the white counts were relatively low, in most cases ranging from 6,400 to 15,700, while two cases had a count of 22,000. However, the patient admitted with acute mastoiditis and nephritis following pneumonia and scarlet fever had a white blood count of 34,000. The blood smears showed a low polymorphonuclear and high lymphocytic count. One blood smear showed 22.0 per cent polymorphonuclear leucocytes, 74.0 per cent

lymphocytes and 4.0 per cent monocytes. Most cases in which definite granulations were found in the mastoid cavities had an eosinophilia ranging from 2.0 to 6.0 per cent.

The majority of cultures showed beta hemolytic streptococci and laboratory tests proved they were sensitive to penicillin in vitro, yet they failed to respond when treated with this agent. This tends to show that the dosages were insufficient to cure the disease, but merely masked the clinical picture. The other cultures were chiefly staphylococcus aureus coagulase positive, and sensitivity tests showed most strains were resistant to penicillin in vitro. Therefore it is reasonable to conclude that, in these cases, good drainage by a myringotomy early in the course of the middle ear infection perhaps offered the best chance of avoiding mastoiditis.

OPERATIVE FINDINGS

In many infants a definite perforation in the mastoid cortex was found. The patients who received the least amount of chemotherapy seemed to show more fluid pus in the mastoid cells, while those who had several spontaneous ruptures and were treated with repeated small doses of drugs contained considerable granulations in the mastoid antra and middle ear cavities. The cell partitions were either soft and decalcified or completely broken down.

MICROSCOPIC FINDINGS

In eight cases specimens removed at mastoidectomy were submitted for microscopic examination. One showed findings quite characteristic of eosinophilic granuloma of bone. It is interesting to note that this patient also had a similar condition of the skin. All the other specimens showed varying stages of chronic infection, some with acute granulation tissue, others with a more chronic process characterized by a dense lymphocytic infiltration.

COMMENT

The obvious purpose of the routine use of chemotherapeutic and antibiotic agents in the treatment of early acute otitis media is to prevent mastoid involvement. The data presented appear to demonstrate the failure of these drugs to prevent mastoiditis and the need for mastoidectomy when used without the benefit of drainage by myringotomy.

SUMMARY AND CONCLUSIONS

The treatment of acute suppurative otitis media with particular reference to the relative merits of chemotherapy and myringotomy in avoiding surgical mastoiditis has been discussed.

The basis of the discussion consisted of clinical, laboratory and operative findings in a series of 30 patients admitted to the Ear House Service of the Massachusetts Eye and Ear Infirmary with acute mastoiditis requiring mastoidectomy, and another series of 45 patients with acute suppurative otitis media treated in the out-patient department by myringotomy.

These two series of cases were observed during the months of April, May, June and July, 1948.

All of the out-patients treated by myringotomy recovered without resort to mastoidectomy. Of the 30 patients admitted to the House Service with acute mastoiditis, all but two had received chemotherapy prior to admission to the hospital but not one of them had had the benefit of a paracentesis. Thirty-six mastoidectomies were required to eradicate the disease in this series of patients.

The pertinent literature dealing with the relative merits of chemotherapy and myringotomy in avoiding surgical mastoiditis is reviewed and discussed.

It is concluded that:

1. The low incidence of surgical mastoiditis in the past few years is to be accounted for by the low incidence of acute suppurative otitis media rather than the result of the routine use of chemotherapeutic and antibiotic agents in this disease.

2. The role of chemotherapy in preventing surgical mastoidectomy is, at best, secondary or adjuvant.

3. Chemotherapy was unavailing in the prevention of surgical mastoiditis in the series of 30 cases treated without resort to myringotomy.

4. Myringotomy prevented surgical mastoidectomy in 45 patients with acute suppurative otitis media observed during the same period of time.

5. In the opinion of the writer this experience, supported by the experience of others reported in the literature makes it abundantly clear that myringotomy is an absolute requirement in the early treatment of acute suppurative otitis media whether or not the use of chemotherapeutic or antibiotic agents are contemplated.

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LVII

FORTY QUESTIONS IN OTOLARYNGOLOGY

(SUGGESTION BOX FOR YOUNG INVESTIGATORS)

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ST. LOUIS, MO.

A fool can pose questions wise men cannot answer.

Fortunately for me, I have been cast today in the former role, and it should be no arduous task to propound some thirty or forty questions for your solution.

To be sure, answers have been forthcoming to most if not all of my questions, for the medical man through the ages must have an answer to everything, no matter how implausible. Furthermore, I am convinced that some of our most widely accepted "truths" will be modified in the future, as they have in the past. Observes Wright, "Some of the passages in the writings of the Aesclepiadae seem ridiculous to us, but we should keep constantly in mind the charity which our successors in their histories will have to extend to the productions of our own times. Indeed, in looking over the various commentaries on Hippocrates from Galen's time to our own, it is curious and not a little amusing to observe how careful each critic is to point out the errors Hippocrates committed in not being in accord with the doctrines of the critic's own time, which are now as obsolete as those of Hippocrates."

To just what degree the following problems may have approached solution is apt to be a matter of personal opinion, depending upon whether one finds acceptable or not the best investigations to date. Let us begin with the nose:

No. 1. What is the nature of the sense of smell? Publications on this subject, which between 1850 and 1900 reached to almost 900, have dwindled to a mere handful since the turn of the century now half-gone. One reads of sporadic experiments with insects, but they do not improve materially upon those of Fabre, and it is still an open question whether the olfactory function is chemical in

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nature, or the result of vibrational resonance, or neither, or possibly a little of both.

No. 2. The function of the sinuses. Textbooks still mention these casually as "adding resonance to the voice," "heating inspired air," "moistening inspired air," "redistributing air" (whatever that may mean), and "lightening the skull." Two of these apply in an unimportant degree, the rest can be demonstrated as false. That sinuses serve in keeping the heads of terrestrial animals afloat when they are forced to take to the water, that they act as insulators against heat and cold, and that they occur incidentally during the repositioning of facial bones after ossification has taken place: all of these are true, but may be incidental to some more fundamental purpose.

No. 3. Origin of sinuses. Are they the result of a primordial urge of the nasal mucosa to invade bone as has been suggested? Probably not. Absence of osteoclasts makes it unlikely that they result from a hollowing-out of bone such as has been described in the so-called bony cysts of the turbinates. To say that they are the result of the natural processes of growth explains nothing. It was "known" until very recently that their early development can be stunted by infection, as noted by Wittmaack in the case of the mastoid cells. Now this is seriously questioned.

No. 4. The nature of ostia—why are they present and why so small?

Obviously it is necessary to have some communication between the rigid sinus cavities and the atmospheric air in order to prevent pressure disturbances, and presumably the smallest possible opening is desirable to render the cavity as nearly closed as possible, and still to permit interchange of air. This latter assumption gains weight from the fact that in the dog's frontal sinus, for example, there is a thin lamina, a partial septum, rendering the inlet more tortuous, and reducing the free circulation of air.

It is unexplained also why the cilia change their direction of beat in the region of the sphenoidal ostium during the formation of the sinus, so that in the upper portion of the cavity ciliary beat will not be away from the ostium, as it would have been without this change of direction.

No. 5. Vacuum in the sinuses. There are several theories for this. First the absorption of air, which is difficult to prove unless a *specific* absorption takes place, which has not been demonstrated. The normal interchange of gases should result in a slightly positive, instead of negative, change in pressure. It is possible that the swelling

of the mucosa may drive out the air, and that the region of the ostium may remain swollen and closed, while there is a recession of the edema elsewhere. This is logical but so far unproven. Hilding has shown further that the ciliary propulsion of thick mucus, out through an ostium, may occur faster than it can be replenished by the glands and thus produce negative pressure.

No. 6. What is a sinus headache? Presumably the result of tension produced by a vacuum, but experiments suggest that the sinus lining is not sensitive except in the neighborhood of the ostium. Whether this applies to an inflamed mucosa, as well as a normal one, has not been made quite clear. There is evidence that a positive pressure may exist in an obstructed sinus during headache.

No. 7. There is much that remains unknown regarding nasal cilia. Why do they beat, always, in one direction? What makes them beat at all? It has been shown that in some of the lower animals continuity of wave propagation is a nerve function. In the human, however, no continuous waves occur and since cilia may persist in beating upon a single cell, or even a cell *fragment* after that cell has been teased away from the the general mass in a culture medium, the beat impulse must be inherent in the cell. It remains unexplained also why the ciliary beat is always toward the pharynx and why, after an injury or denudation, the direction of beat in the new tissue conforms to that in the old. Also, why does normal regeneration sometimes occur after a complete stripping of the sinus wall and at other times fail completely?

No. 8. Congenital choanal atresia. Why is it that in this condition although obstruction persists for many years, infection and hyperplasia of the nasal structures and the eustachian tube do not take place, although these are characteristic of obstruction brought about by other agencies which have their beginnings some time after birth?

No. 9. What is the nature of a sneeze? Is it the cause or the effect of sudden changes in the engorgment of the turbinates? The accompanying "goose flesh," which pervades other portions of the body the same time, would indicate the latter; or would it?

No. 10. What is the nature of the superposition of stimuli by which a half-baked sneeze can be brought to fruition? This seems to be effective only if the added stimulus is one of light. A similar reaction does not follow strong taste stimuli such as salt on the tongue, or tactile ones such as a finger prick; in fact, sharp noise may serve to sidetrack the half-developed sneeze.

I know of a case, however, in which moderate tactile stimulation with the finger or with a hair brush at the hairline in the frontal region will promptly and invariably produce sneezing.

No. 11. Cerebrospinal rhinorrhea. What are the causes and the nature of the spontaneous type? Why is it likely to be intermittent? What prevents prompt infection of the meninges?

No. 12. What are the effects of the endocrines on the respiraatory system? There is evidence that the pituitary and the adrenal glands influence the status of the nasal mucosa in some degree and under some circumstances. It has been shown that the thyroid hormone, and especially the lack of it, produces altered tone and an increased sensitivity to infection and histamine. The exact mechanism of these phenomena has not been explained.

No. 13. The intimate connection between the nose and the genital organs has received not infrequent attention, but the nature of these relations remains vague. So-called vicarious menstruation from the nose has latterly been interpreted as merely nonspecific bleeding, resulting from the general congestion. Vascular changes in the nose accompanying genital tumescence and detumescence may fall into the same category. The recurrence of polyps during successive pregnancies also requires explanation. In the late '90s Fliess wrote a monograph on the relationship of the nose and the genital tract; however, he allowed himself to be caried away by his subject to a point which made it difficult to distinguish between fact and fancy, thus greatly impairing the value of his work.

No. 14. Atrophic rhinitis. Practically everything about this disagreeable disease requires explaining; its cause, the nature of the atrophy and above all the treatment. This last falls into two methods of approach, the mechanical reduction of the air spaces and the attempted regeneration of the mucosa through the administration of the female hormone. The first is only partly successful; the second, based on purely theoretical grounds, even less so.

No. 15. Idiosyncracies to drugs. In rhinological practices the hypersensitivity to cocaine and pontocaine is probably the most serious. Idiosyncracies to atropine, codeine, aspirin and morphine can be troublesome. In this connection may be mentioned also that terrifying but quite hypothetical condition formerly designated as "status lymphaticus."

No. 16. The relationship of sinusitis to retrobulbar neuritis. Considerable controversy has arisen from time to time as to whether or not such relationship exists. It has been both affirmed and denied

by observers of more than average ability. A direct connection between the nasal sinuses and the optic nerve has been described. Usually the effect of nasal operations upon the condition have been attributed to the resulting decompression. Leon White, who devoted much of his life and work to the study of the relationship between the optic nerve and its foramen, and was convinced that the pressure of a small foramen could be responsible for the condition, was later obliged to concede (and had the courage to do so) that he was mistaken.

No. 17. Sinuses and mental disease. Some fifteen years ago two English investigators, Pickworth and Graves, through a series of published papers stimulated considerable interest in the possible relationship between infected sinuses and the various delusional states. Apparently little was done by others, either to confirm or to disprove their contentions, and the problem, still unsolved, seems to have disappeared from the medical prints.

No. 18. The sinuses and rheumatism. The position of the sinuses in the rôle of foci of infection has not been well established, although most writers regard it as relatively unimportant. Writers are agreed that where sinus infection exists, in cases of rheumatism and other similar remote affections, it should surely be eradicated. They are less in agreement regarding the results obtained.

No. 19. Juvenile nasofibroma. This fearsome tumor requires explanation both as to etiology and location. Springing as it does from a region not especially vascular, its structure also requires explaining. The details of its management are likewise controversial especially regarding the value of irradiation either with or without subsequent surgery, the method of removal, and the necessity of preliminary carotid artery ligation.

No. 20. Malignant granuloma. This uniformly fatal lesion defies classification and treatment. The most recent comprehensive review of the condition is probably that which Lierle published some three years ago. The prognosis is still hopeless.

No. 21. Familial telangiectasis. This is another of the distressing and sometimes fatal conditions of undetermined cause and development. This hereditary deficiency in the components of the vascular walls is probably best managed at the present writing by the persistent, painstaking and systematic application of high-frequency desiccation as described by Figi, although the treatment cannot be said to be universally accepted.

No. 22. The common cold. Despite the antihistamine bubble which has burst damply in our faces, the common cold continues an unsolved problem. We do not know what it is, how to prevent it, or how to cure it.

No. 23. The function of the tonsil. A generation or so ago, the question of tonsil function was uppermost in many minds. It was suggested that the tonsil is a hemopoietic organ; that it is a filter against bacteria; that it has an internal secretion; that it elaborates antibodies from absorbed bacteria. Wright discovered, for example, that bacteria which did not ordinarily penetrate the tonsil could be made to do so by coating them with butter, an interesting observation which aroused only passing attention.

No. 24. The action of the epiglottis. In earlier days it was assumed without much actual investigation that the epiglottis was a trap door to prevent the entrance of food and other solid material down the glottic chink. Later it was authoritatively stated that the epiglottis was actually a rigid structure which protected the glottis by diverting food and drink to the sides and into the pyriform fossae, and it was pointed out that no muscles exist which could turn down the epiglottis as a trap door.

Only last spring, in San Francisco, a strip of radiological motion pictures was exhibited by Saunders and his associates which seemed to show that the larynx rising during deglutition forced the epiglottis against the tongue in such a manner as to tilt it backward to cover the glottis. Some of the foremost endoscopists are still not convinced by the demonstration, and the action of the epiglottis remains among the unsolved problems.

No. 25. The purpose of a yawn. The nature of this interesting phenomenon is not only unexplained in most texts on physiology, but is completely ignored. It is not to be found in the indexes of any of the volumes at my disposal. The dictionary shrugs it off as "an involuntary deep inspiration with the mouth open:" a feeble definition, if I ever saw one.

It would appear at first consideration that this deep inspiratory effort might be for the purpose of increasing the oxygen intake, but since it lasts three or four times as long as a normal inspiration it would seem to defeat its own purpose.

No. 26. The eustachian orifice and its environs. This problem, which seemed completely solved for many years until the exigencies of aerial warfare once more focused attention upon it, seems still to require some investigation. The causes of so-called aero-otitis

media and the attempt to combat it by irradiation of its lymphoid constituents evoked a more careful study of the distribution of these tissues. Some, as yet unpublished, work by Semenov delves further into the unknowns of this region.

No. 27. Lingual pain and the menopause. The onset of unexplained glossopharyngeal neuralgia at and after the menopause requires explanation as does the self-limiting nature of this excruciating pain.

No. 28. Parageusia in the aging. Probably closely associated with the last category of symptoms is the bitter taste complained of by women in the fifth and sixth decades of life. The presence and the self-limitation of the symptom require explanation, as does the fact that it occurs almost exclusively in women.

No. 29. The nature of the passage of infection between the pharynx and the trachea. It has always been taken more or less for granted that infectious material from the upper respiratory tract passed through the lower, directly, via the chink of the glottis. However, large masses of such material must undoubtedly set up a bechic reflex to expel them, and microscopic quantities deposited in the upper trachea would inevitably be raised to the glottis by the action of the cilia in that region. Furthermore, we are taught that no lymphatics traverse the vocal cords. A discussion of the question invariably brings the answer "well it occurs during sleep," but if the bechic reflex is lulled to rest at such times the breathing is also quiet and the cilia are still active.

It might be pointed out that while a sudden explosive *expiratory* blast occurs, which we call a cough, there is no comparable inspiratory blast to carry infectious material *into* the trachea. It seems quite likely that infection from the bronchial tree to the nose occurs quite as frequently as the opposite.

The lymphatic connection between the upper respiratory tract and the deeper pulmonary glands has been shown; not so, any connection with the upper trachea.

No. 30. The management of bilateral recurrent paralysis. The multiplicity of methods suggested for the remedy of this condition still leaves it in the category of unsolved problems. The details are many and intricate and so are the articles dealing with them.

No. 31. Papilloma laryngis. Since the cause of this condition is unknown, the management is still empirical and not too satisfactory. Repeated removal is still required. The endocrine ap-

proach, based upon theoretical concepts, is not meeting with the anticipated success.

No. 32. Cancer of the larynx, its origin, its spread, and its treatment. Conferences upon this subject have much the flavor of conferences held ten (and twenty) years ago: irradiation, what kind, and how much? Surgery, what kind, when, and how extensive.

No. 33. Tobacco and pulmonary cancer. The connection is thus far inferential. That there has been a sudden increase in bronchial and pulmonary cancer in the last three decades is a fact. That cigarette smoking has increased enormously in the same time is a fact. That the increase in bronchial cancer occurs in nonsmokers as well as smokers is a fact. That the relationship between the two has not been established is regrettably also a fact.

No. 34. The nature of hearing. The earlier theories of hearing were based largely upon models of the cochlear apparatus, and on hearing tests. The discovery of the Wever-Bray phenomenon permitted and stimulated a new type of research. Many investigators, among whom the names of Lorente de No, Davis, and Hallpike spring to mind, are busy with the problem.

No. 35. Absolute pitch. Why some persons have the ability to recognize the pitch of a tone as most of us recognize a color remains unexplained. Colorblindness is the exception. Pitchdeafness the rule.

No. 36. Diplacusis. When we solve the problem of sound perception then the nature of diplacusis will be better understood. It is possible that the observation of conditions in diplacusis may help to establish and confirm theories of hearing.

No. 37. Musical interpretation centers. The complex mechanism of musical perception and conception is still a closed book. I know of at least one case in which a cerebral accident deprived an individual of his ability to read notes while he continued to recognize melodies by ear and also retained his sense of absolute pitch.

No. 38. The cause of tinnitus. Once we learn to recognize and to differentiate the causes of this distressing symptom we may be able to prevent it and to abandon our present desperate remedy, the euthanasia of the eighth nerve.

No. 39. The interpretation of high-tone loss and other similar acoustical phenomena. The acoustics of the skull are complicated and variable. It is understandable that a similar high-tone loss may occur in such disparate conditions as nerve degeneration and fluid

in the middle ear on this ground. Demonstration of the process is still wanting.

No. 40. Otitis externa diffusa, the nature of cerumen, and aberrations of the skin of the canal and its glands are among the problems brought into prominence by the war in the Pacific. The work of Senturia promises an early, and we hope effective, solution.

No. 41. The nature of otosclerosis. Concerning this disease we are not without theories—emphatically expressed—some logical and some fantastic, but we are still in the dark as to what it is, how to prevent it, or how to stop its progress. The importance of the problem is indicated by the fact that an organized group exists for its study.

No. 42. The final, burning, sixty-four thousand dollar question: how to keep that new oval window open. Not long ago a group of otologists in South America were discussing the work of an investigator.

"I have spoken with him," said one of the colleagues, "he has the final answer."

Eyebrows went up.

"Do you believe that?" they asked.

"Well," he replied, "he ought to know a final answer when he sees it. This is his *fifth* final answer."

* * *

The fool has asked his questions; let the wise men take over.

BEAUMONT MEDICAL BUILDING.

LVIII

SURGERY IN MÉNIÈRE'S DISEASE

A NEW OPERATION WHICH PRESERVES THE LABYRINTH

REPORT OF CASES

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NEW YORK, N. Y.

The present surgical treatment of unilateral Ménière's disease consists mainly in destroying the affected labyrinth with consequent complete loss of vestibular and cochlear functions. Destructive labyrinthotomy has largely replaced the more formidable intracranial section of the eighth nerve as well as incision and drainage of the saccus endolymphaticus.

The most disturbing symptom of Ménière's disease is recurrent aural vertigo—sudden violent paroxyms of vertigo and falling, followed by nausea and vomiting. The vertigo varies in intensity and duration. The deafness, tinnitus, hyperacusis, distortion of sound, diplacusis and caloric hypo-excitability occur in varying combinations and varying degrees. The deafness, which is subject to sudden fluctuations, involves the low and high frequencies, with diminished bone conduction. The tinnitus is usually a low-pitched, roaring sea-shell noise, with a second, less disturbing, high-pitched ring. At times the tinnitus is like the hum of noisy machinery. Extreme general distress and acute discomfort in the affected ear result from loud sounds (subway trains, passing trucks, rattle of table silver or crockery, or high-pitched radio). The patient often hears the same tone at a different level in the two ears. Caloric stimulation of the affected labyrinth gives much less than normal responses as to vertigo, nystagmus, and past-pointing, especially of the vertical canals.[8] Sometimes caloric hypo-excitability is present on both sides, but more marked on the manifestly affected side.

The destructive operations on the labyrinth have been eminently successful in eliminating the attacks of vertigo, but much less successful in eliminating the tinnitus and the severe sense of pressure and fullness in and about the ear and head. Following labyrinthotomy, the hearing is practically always totally destroyed. To sac-

From the Department of Otolaryngology, the Mount Sinai Hospital, New York City, N. Y.

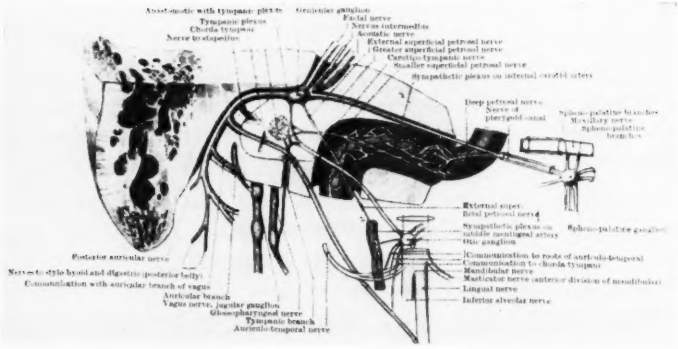


Fig. 1.—Connections of the facial nerve in the temporal bone. (Cunningham) Note the branch of the nervus intermedius communicating with the vestibular portion of the eighth nerve in the internal acoustic canal.

rifice the hearing involves a risk, since in ten per cent or more of the cases[9] the disease is, or will become, bilateral. Hence, the ideal surgical procedure would eliminate the vertigo and preserve the hearing.

The pathology of Ménière's disease has recently been demonstrated histologically[10]—an endolymphatic fluid distention of the cochlear and vestibular portions of the membranous labyrinth. When this hydropic distention involves the cochlea alone[11] (Case 3), as it may, the signs and symptoms are deafness, tinnitus, hyperacusis, diplacusis, and distortion of sound. When the cochlear hydrops spreads to the utricle (static labyrinth), then attacks of vertigo supervene. Both cochlear and vestibular portions are usually involved. Though the mechanism of the hydrops is not completely understood, the most accepted explanation is that imbalance of the autonomic nervous system[12] or a sort of allergy[13] gives rise to a vaso-spastic-atonic state of the capillary loops to the endolymphatic labyrinth, resulting in fluid transudation. As this excessive fluid accumulates, the endolymphatic tension increases until the signs and symptoms of Ménière's disease appear (like glaucoma). Therefore, operations on the autonomic nervous system (sympathetic denervation, i.e., operations on the stellate ganglion, cervical sympathetics, stripping the common carotid arteries)[14] hold some promise in eliminating or reducing capillary spasm, thereby reducing the endolymphatic hypertension. Continuous intravenous procaine for the relief of myoneural capillary spasm, together with vaso-dilator agents, also offer symptomatic relief.[15, 16]

There is still before us the challenge to develop otology so that, for Ménière's disease, we may ultimately avoid the necessity to destroy such an important portion of the nervous system as the labyrinth. Experience gained in utilizing the chorda tympani nerve to cover the fenestra in the fenestration operation[17] led to the surgical procedure here described in cases of Ménière's disease, a procedure which leaves the vestibular labyrinth and the cochlea intact.

This procedure is based on a new theory[18] of hearing, the essence of which is that there are two sonic systems in man: one, the more recent cochlea, and, two, the older, primitive sonic system derived from the lateral line organs of aquatic vertebrates. In these primitive animals the seventh and ninth cranial nerves carry the sonic and equilibratory impulses to the brain stem. The chorda tympani (seventh cranial) and tympanic plexus-Jacobson's nerve (ninth cranial) are derivatives of this primitive system and both enter the geniculate ganglion. The chorda tympani nerve at the geniculate ganglion is a continuation of the nervus intermedius (nerve of Wrisberg), and the nervus intermedius communicates with the vestibular branch of the eighth cranial nerve (the newer sonic system) in the internal auditory canal (Cunningham's Anatomy[19]) (Figs. 1 and 2). Thus the old sonic system (seventh and ninth nerves) is connected with the newer sonic system (eighth nerve) via the geniculate ganglion and via branches of the tympanic plexus-Jacobson's nerve to the oval and round windows. Thus, if and when these primitive sonic and equilibratory nerves seventh (chorda tympani) and ninth (tympanic plexus-Jacobson's nerve) are affected, then impulses arising in the tongue, jaw-joint, teeth, pharynx, tympanum and sinuses could stream toward the geniculate ganglion, thence via the nervus intermedius to the eighth nerve, or through the branches of Jacobson's nerve to the oval and round windows to give rise to capillary spasm, and ultimately endolymphatic hydrops. The clinical picture of Ménière's disease would result.

The purpose of this paper is to describe a simple operation for cases of Ménière's disease based on the above theory. I have carried out this operation on 14 patients whose signs and symptoms suggested a hydrops of the labyrinth. The operation consists merely in sectioning the chorda tympani nerve alone or sectioning both the chorda tympani and the tympanic plexus-Jacobson's nerve.

TECHNIQUE

The operation is performed through an ear speculum in the external auditory canal.[20] Prior cadaver practice is invaluable.[21] Mild

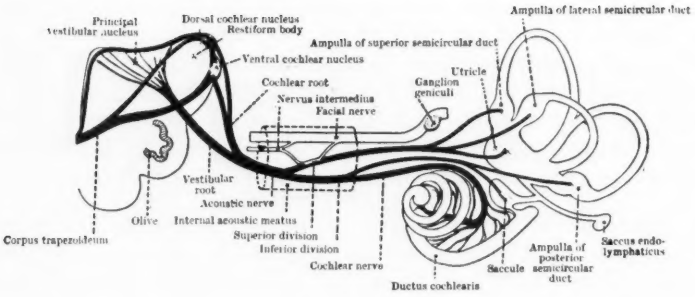


Fig. 2.—Scheme of the origin and distribution of the acoustic nerve. (Cunningham) Note the branch of the nervus intermedius communicating with the vestibular portion of the eighth nerve in the internal acoustic canal.

sedation and local novocaine injection with adrenalin in the membranous canal wall were used to keep bleeding at a minimum. Patients were thus sufficiently alert to note sensations and report them during the operation.

An incision is made through the skin over the bony canal wall (on the right side beginning at nine o'clock and extending to three o'clock, and on the left side beginning at three o'clock and extending to nine o'clock) about five to seven mm external to the drum. The skin is separated from the bone as far as the drum. The drum is then lifted out of its sulcus and reflected upward upon itself like an apron, thus exposing the tympanic cavity. In half the cases, the chorda tympani was immediately visible for about four to six mm close to the edge of the posterior bony canal. In the rest, it was teased out into the tympanum from just behind the bony edge of the canal wall. Before the chorda was sectioned, it was decided to observe its response to electrical stimulation. Accordingly, the intact chorda was stimulated with a Faradic type 120 cycle current. The current characteristics found effective were two to three volts at 0.2-0.3 milliamperes. Long, insulated electrodes were used. In all cases, each chorda stimulation (contact of chorda with electrode) was followed instantly by intense contraction of the entire same side of the face. Some patients reported a marked increment in tinnitus and rotatory vertigo with each stimulation of the chorda. One patient reported cessation of tinnitus with each stimulation. All sensations subsided immediately when the stimulation was stopped, only to recur with each new stimulation.

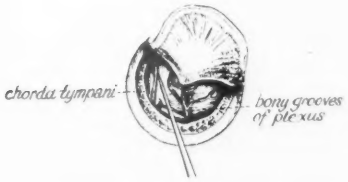


Fig. 3.—Eardrum lifted out of its sulcus and reflected upward upon itself. The chorda tympani nerve can be seen in the grasp of the iris hook ready to be sectioned. The bony grooves of the tympanic plexus of nerves can be seen.

In some cases, the chorda tympani nerve alone, and in others, the chorda tympani and the tympanic plexus were then sectioned. The chorda was sectioned by engaging it in a small iris hook and pulling outward. Jacobson's nerve and its branches were scraped away from the promontory and sectioned with a sharp dental hoe excavator (often Jacobson's nerve and some of its branches are completely encased in bone.)[22, 23] The sharp dental hoe, however, removes the bone-imbedded nerves (Fig. 3).

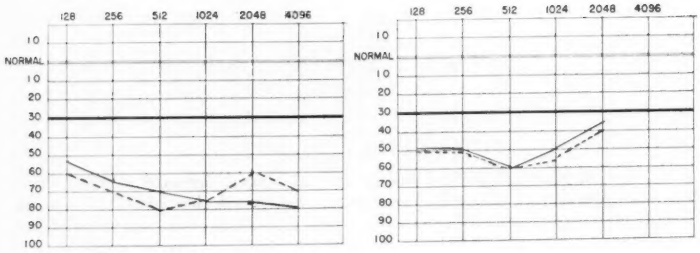
After the chorda was sectioned in the tympanum, the electrical stimulus was applied to each cut end, i.e., to the central end (that portion running centrally to the geniculate) and to the peripheral end (that portion running to the tongue). Stimulation of the central cut end of the chorda gave similar responses to those of the intact chorda, whereas stimulation of the peripheral cut end of the chorda gave none of the above responses. A more detailed report on these experiments is in press.

CASE REPORTS

CASE 1.—F. A., female, aged 60, housewife. Frequent attacks of incapacitating rotatory vertigo, nausea, vomiting, and falling for past four years. Bilateral progressive deafness, worse during vertiginous attacks. Deafness more marked in right ear. Loud, constant humming tinnitus in both ears, worse in right. Caloric tests showed hypoactive responses from the horizontal canals on both sides, worse on right, and no responses from any of the vertical canals. Medical, neurological, laboratory, and allergic findings negative.

December 12, 1949, section of right chorda tympani and Jacobson's nerve-tympanic plexus. Out of bed in 24 hours. No vertigo since operation. Tinnitus much less marked.

CASE 2.—F. P., female, aged 47, domestic. Recurring attacks of rotatory vertigo with nausea, vomiting, and falling past ten years. Constant low-pitched roaring tinnitus, fluctuating deafness and hyperacusis in right ear for past ten years. Hypoactive caloric responses right side. Anxious after attacks and fears crossing the street. Medical, neurological examinations negative. Conservative treatment with histamine, etc., unsuccessful.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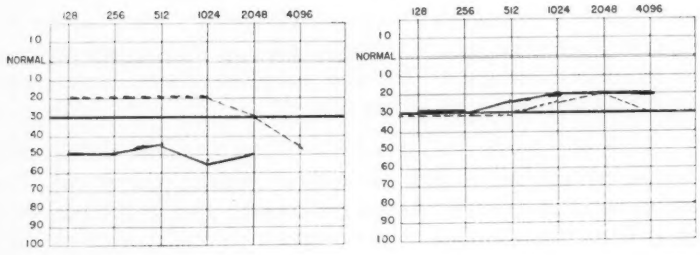
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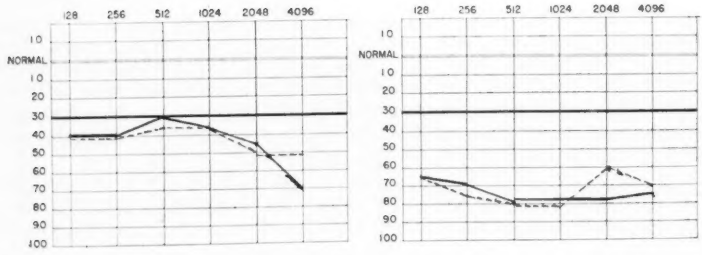
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Fig. 4, Case 1.—F. A. Operation Dec. 12, 1949. No vertigo since operation. Less tinnitus. Solid line indicates pre-operative. Broken line 17 months post-operative.

Fig. 5, Case 2.—F. P. Operation Dec. 20, 1950. No vertigo and no tinnitus since operation. Solid line indicates pre-operative. Broken line 6 months post-operative.

Fig. 6, Case 3.—M. M. Involvement of cochlea alone. Operation April 19, 1949. Excellent hearing persists since operation. Tinnitus less. Solid line is average of many pre-operative tests and broken line the average of many post-operative tests.

Fig. 7, Case 4.—B. G. Operation April 16, 1950. No vertigo since operation. Tinnitus less. Solid line indicates pre-operative. Broken line 9 months post-operative.

Fig. 8, Case 8.—F. R. Operation Nov. 27, 1950. No vertigo since operation. Tinnitus less. Solid line indicates pre-operative. Broken line 6 months post-operative.

Fig. 9, Case 12.—M. S. Operation June 15, 1949. Vertigo persists. Tinnitus same. Solid line indicates pre-operative. Broken line 23 months post-operative. In all fourteen patients operated, the hearing was preserved.

December 20, 1950, section of right chorda only. No vertigo, tinnitus or hyperacusis since operation. (Electrical stimulation of the chorda tympani gave prompt rotatory vertigo and increased tinnitus with each stimulus. The vertigo thus induced was similar to the preoperative vertigo).

CASE 3.—M. M., female, aged 39, business executive. For two years, frequent attacks of sudden deafness in left ear which lasted for a few hours to several days. Low, roaring tinnitus most of the time, but much worse during attacks of sudden deafness. Marked hypercusis, diplacusis, and sound distortion. No vertigo. For six months before operation, the attacks of sudden deafness occurred every two to three weeks and the hearing in the left ear became worse. Vaso-motor rhinitis and history of food allergy with skin eruptions. Treatment unsuccessful.

April 14, 1949, section of left chorda tympani and Jacobson's nerve-tympanic plexus. Since operation the hearing has returned to a high level and has not receded. The low roaring tinnitus is absent, but there is present a high-pitched tinnitus which is not annoying.

CASE 4.—B. G., female, aged 48, housewife. Attacks of rotatory vertigo nausea, and vomiting once or twice a week for past year. Fluctuating deafness, constant roaring tinnitus, hyperacusis, and stuffiness in left ear for one year with associated left-sided headache. For five years there has been slight diminution in hearing. Medical, neurological, psychiatric, allergy consultations in O.P.D. and hospital negative, lab findings negative.

April 16, 1950, section of left chorda tympani nerve and Jacobson's nerve-tympanic plexus. Since operation, no vertigo. Tinnitus, headache and hyperacusis less marked.

CASE 5.—A. M., male, aged 53, physician. For 14 years, mild attacks of rotatory vertigo with roaring tinnitus and fluctuating deafness in the right ear. For past two years, attacks of vertigo, nausea, vomiting and headache more intense and frequent. All conservative therapy failed.

October 19, 1950, section of right chorda tympani and Jacobson's nerve-tympanic plexus. Since operation, no attacks of vertigo. Tinnitus almost gone. Headache persists.

CASE 6.—J. W., male, aged 30, radio announcer. Five attacks of rotatory vertigo, nausea and vomiting in past year. Low, hissing tinnitus, fluctuating deafness, and hyperacusis on right side for 14 months. Medical, neurological, allergic examinations negative.

June 19, 1950, section of right chorda tympani and Jacobson's nerve-tympanic plexus. For four months following the operation there was no vertigo. Since then, the vertigo has returned, though less frequent and less intense. The tinnitus, fluctuating deafness and hyperacusis persisted.

CASE 7.—R. W., female, aged 33, housewife. Attacks of rotatory vertigo falling the patient once a month for past 18 months. Progressive deafness and feeling of fullness in left ear past ten years. Low-pitched roaring and hissing tinnitus and hyperacusis in left ear for several years. Medical, neurological examinations negative.

November 22, 1950, section of left chorda tympani and Jacobson's nerve-tympanic plexus. Out of bed in 24 hours. Since operation, no vertigo. Roaring tinnitus gone. Slight high-pitched tinnitus, but not annoying.

CASE 8.—F. R., female, aged 54, machine operator. Weekly attacks of rotatory vertigo, nausea, vomiting and falling past six months. Constant low roaring tinnitus, fluctuating deafness and hyperacusis in left ear for past two years, with

occasional vertigo. Hypoactive caloric responses on left side. Diplacusis. Mild diabetic, otherwise entirely negative physical, neurological and psychiatric findings.

November 27, 1950, section of left chorda tympani only. Out of bed in 24 hours. Since operation, no vertigo or low, roaring tinnitus. There is present a high-pitched, ringing tinnitus, not annoying.

CASE 9.—L. B., male, aged 48, shipper. Attacks of incapacitating rotatory vertigo past three years. Past six months, attacks associated with nausea and vomiting, so severe he could not work steadily. Low, roaring tinnitus and fluctuating deafness in left ear for three years. Left labyrinth hypoactive. Hyperacusis and diplacusis. Medical, allergic treatment unsuccessful.

September 14, 1950, section of left chorda tympani and Jacobson's nerve-tympanic plexus. Out of bed in 24 hours. No vertigo since operation. Tinnitus persists.

CASE 10.—I. P., female, aged 70, housewife. Frequent attacks of rotatory vertigo, nausea and vomiting past year. Confined to bed past two months because of vertigo. Hissing tinnitus and fluctuating deafness in left ear for four years. Conservative treatment failed. Medical, neurological, psychiatric and allergic studies negative.

December 28, 1950, section of left chorda tympani alone. Since operation, no vertigo. Tinnitus same.

CASE 11.—J. U., male, aged 45, machine cutter. Attacks of rotatory vertigo, nausea and vomiting twice a week for past six months. Unable and afraid to work at machine. Mild attacks for two years. Continuous loud hissing tinnitus and marked deafness in left ear. Deafness fluctuates. Hyperacusis and caloric hypoexcitability on left side. Diplacusis.

January 10, 1951, section of left chorda tympani nerve alone. Since operation, no vertigo or hissing tinnitus. There is a slight high ringing tinnitus since operation, inconstant and not annoying. Deafness still fluctuates, but hyperacusis almost gone.

CASE 12.—M. S., female, aged 28, housewife. Attacks of rotatory vertigo, falling, nausea and vomiting every week for past nine months. Constant, loud, hissing tinnitus, hyperacusis, and fluctuating deafness on left side for past year. Medical, neurological, and allergy studies negative. Caloric tests normal on both sides.

June 15, 1949, section of left chorda tympani nerve and Jacobson's nerve-tympanic plexus. No vertigo until October 19, 1949. Patient pregnant at this time. The vertigo and tinnitus have persisted to this date, though less severe.

CASE 13.—G. R., male, aged 49, office worker. For past 15 years attacks of vertigo, nausea, vomiting, with fluctuating deafness. Loud, roaring tinnitus, greater in right ear than left. An arrested pulmonary tuberculosis. Otherwise medical, neurological examinations negative.

December 12, 1950, section of right chorda tympani nerve only. No vertigo since operation, but tinnitus is about the same.

CASE 14.—M. E., male, aged 49, broker. For past 11 months, monthly attacks of rotatory vertigo, left-sided headache, nausea, palpitation, sweating and vomiting. Low, roaring, constant tinnitus, hyperacusis and fluctuating deafness in left ear. Caloric gave less active responses from left side than from right. Medical, neurological, allergy examinations negative.

January 18, 1950, section of left chorda tympani and Jacobson's nerve-tympanic plexus. No vertigo since operation. Tinnitus less marked, but disabling headache and pressure in left side of head persist.

SUMMARY

1. A new surgical procedure for Ménière's disease is described which preserves the labyrinth.

2. Fourteen patients were operated upon by the technique described. Eleven of these have had no vertigo since operation. In two patients the vertigo has persisted. In one patient (Case 3) with Ménière's disease limited to the cochlea, the hearing has returned to a very high level, and has not dropped since the operation.

3. In one patient the tinnitus has disappeared altogether. In seven the tinnitus has diminished so as not to be annoying, whereas in six it has remained unaffected by the operation.

4. In none of the cases operated upon was the hearing destroyed.

5. This procedure is simple, requires two or three days of hospitalization, and is performed under local anesthesia.

6. This operation can safely be performed on both ears in cases of bilateral Ménière's disease.

7. This conservative procedure should be tried before resorting to the radical destructive labyrinthotomy.

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LIX

A COLD VAPOR APPARATUS FOR THE TREATMENT OF ACUTE LARYNGOTRACHEITIS

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This report demonstrates the applicability of a simple, inexpensive method for providing humidity, controlled temperature, and a high concentration of oxygen in the treatment of acute infectious laryngotracheitis.

Acute laryngotracheitis is an infection of the larynx, trachea and bronchial tree, characterized by inflammation and edema of the larynx, and a thick, adherent, purulent secretion which obstructs the lower respiratory passages. The symptoms of this disease are notoriously rapid in their onset.

A typical symptom complex is as follows: a child, well 15 hours prior to admission to the hospital, suddenly develops an elevation of temperature; becomes hoarse; has stridulous and croupy respirations; develops dyspnea, and eventually prostration and toxemia. Severe retraction of the suprasternal, infrasternal areas and the intercostal spaces with marked "accordion-like" movements of the thoracic cage present a picture which is terrifying to the parents and of grave concern to the attending physician.

When the patient is admitted to the hospital he is immediately placed, for comfort, in a specially prepared steam room. This is extremely important because eight to 12 hours are required for the specific medications to effectively combat the infection causing the acute laryngotracheal-bronchial stenosis.

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Copeland[1] stated that marked relief was afforded to his patients with this infection by the use of an excessively humid warm atmosphere. Quoting Brenneman[2] he maintained that the room or tent may become depressingly warm if steam alone is employed for the humidification. A humidifier with added oxygen made some of his patients more comfortable. Rabe[3] studied 347 patients with infectious croup who were admitted to the New Haven Hospital from 1937-1946, inclusive. In addition to prompt chemotherapy he considered it best to place all the patients in a steam room and watch them carefully. Two hundred ninety-three patients with croup were admitted to the Charles V. Chapin Hospital in the years 1935-1948. There were 14 deaths in this series, eight of whom had received tracheotomies.

At the Charles V. Chapin Hospital, therapy for acute laryngotracheitis is divided into two parts, each equally important to the care of the patient. The first part involves specific medication. Because this is frequently a mixed infection the patient is immediately started on combined streptomycin and penicillin therapy. The former combats the gram positive organisms; the latter controls the gram negative organisms. In severe cases in which a virus is suspected the administration of aureomycin is occasionally considered. The streptomycin is omitted after 48 hours, and the penicillin is discontinued when the condition of the patient no longer warrants continuation of the drug. Blood cultures and nose and throat cultures are performed routinely on admission. Discs sensitivity tests are performed and further medication is governed by the results reported from the laboratory concerning the in-vitro sensitivity tests.

The second part of the program for treating acute laryngotracheitis is the use of moist, warm steam. Patients are placed in the "croup room" in which the humidity is controlled by steam, piped directly from the central heating plant; the comfort of the patient being the primary purpose. A relative humidity of 60-70%, a temperature of 65° F. are desired in the steam room. Unfortunately this is frequently not obtained. The room may become overbearingly warm with the temperature rising to 90° F., and the humidity stubbornly refusing to rise above 50% even though the ceiling and windows are dripping wet and the special nurse and patient are perspiring profusely in an atmosphere of fine moist rain. Our experience has been the experience of others.

It has been observed that children desperately ill at home frequently improve by the time they arrive at the hospital. The ride to the hospital in the cool, moist night air produces a temporary

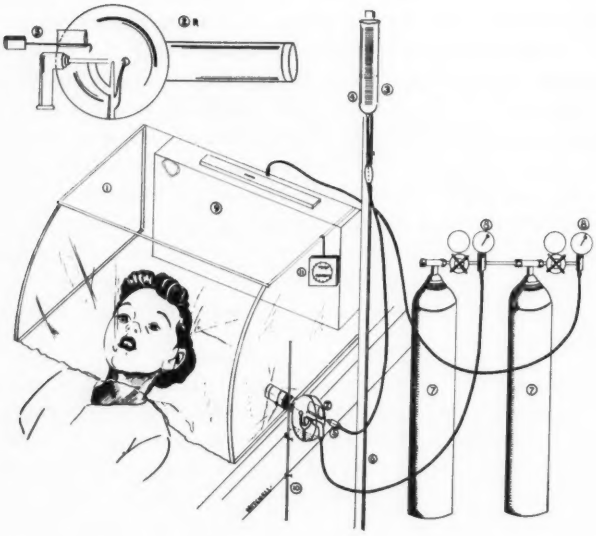

Fig. 1.—Simple apparatus for vapor therapy. 1. Croup tent (Burgess box); 2. nebulizer (Vaponefrin); 3. wetting agent reservoir with tubing; 4. wetting agent solution; 5. curved No. 21 gauge needle; 6. pole for cold vapor reservoir; 7. oxygen cylinder; 8. flow meter; 9. ice chamber; 10. stand for nebulizer; 11. hygrometer.

remission of the obstructing symptoms. This improvement is swiftly negated by the dry warmth of the hospital ward room.

Cool moist air is more comforting to these patients than hot arid air, and is essential in eliminating the dryness of the respiratory mucous membrane. A relative humidity of nearly 90% must be maintained in the steam room if the tracheobronchial mucous membrane is to be kept moist.

A wetting agent is a substance which decreases the surface tension of water. When added to water a wetting agent will cause the water to give a higher degree of wetting against a contiguous phase than would be obtained with pure water. Van Antwerpen[4] published a list of nearly 300 commercially available wetting agents. Some of them are used in dentifrices and hydrophilic ointments.

Sodium lauryl sulfate U.S.P. (Duponol-C$^{(R)}$) is a mixture of sodium alkyl sulfates consisting principally of lauryl sulfate. One gram of sodium lauryl sulfate dissolves in 10 cc. of water forming

an opalescent solution. The crystals are small and white with a slight characteristic odor. It is approved by the Pure Foods and Drugs Commissioner, and in concentration of 0.1% is apparently non-toxic to the human. We tested several wetting agents and chose 0.1% sodium lauryl sulfate U.S.P. for the treatment of acute infectious laryngotracheitis in the belief that it would keep the tracheobronchial tree moist and free from dry, tenacious secretions.

DESCRIPTION AND USE OF THE APPARATUS

Figure 1 depicts the apparatus used in treating ten patients with acute laryngotracheitis. Sodium lauryl sulfate solution, 0.1%, was vaporized to provide the patient a cool, moist temperature and controlled atmosphere. A Burgess box served as a croup tent. Into this confined area, cold vapor was produced by nebulizing sodium lauryl sulfate solution, 0.1%, under constant pressure of five pounds. Positive pressure can be obtained by several methods; compressed air, oxygen, etc. We used an oxygen cylinder (244 cu. ft.) and a vaponefrin(R) nebulizer. Particle size of the cold vapor varied from two to five μ. It was found that four liters of oxygen per minute were adequate to vaporize three to five minims of solution and quickly produce a humidity of 70% and a temperature of 65° F. The use of oxygen as the pressure agent maintained a concentration of 40% oxygen in the "croup tent," but additional oxygen was run into the Burgess box at four to 6 liters per minute.

It was found that in a short time the humidity inside the tent rose to 90%. Lowering the rate of oxygen flow to two liters per minute maintained the humidity between 60-70% and the temperature between 60-65° F., optimum in treatment.

REPORT OF A CASE

The record of one of the first patients treated in this manner is briefly summarized.

L. C., 17 months female with difficulty in breathing, fever and cough of 24 hours duration, progress worse. Malaise, stupor and cyanosis increased prior to hospitalization. On admission the temperature was 102.4° F., apical pulse 140 per minute, respiration was 50 per minute. The child was in marked respiratory distress; had a stridulous cough; cyanosis of the lips, finger tips and toes, and a peculiar mottling of the forearms, hands, lower legs and feet. Nasal mucous membranes, pharynx, tonsils, and right ear drum were injected. Alae nasae were flaring. There were marked costal and sternal retractions; prominent coarse rhonchi without rales in both lungs.

At 9:15 p.m. the child was placed in the "croup tent" and 0.1% cold vapor administered. Forty-five minutes after initiation of the cold vapor the tent registered a humidity of 70% and a temperature of 70° F. At 11:30 p.m. the patient's temperature, pulse and respirations were 100.6° F., 96 per minute, 40 per minute. The tent registered 72% humidity and a temperature of 63° F. The child was sleeping comfortable, still retracting mildly; color was much im-

proved the next morning. At 8:00 a.m. the patient's temperature, pulse and respirations were 99.8° F., 100 per minute and 30 per minute. In the tent the humidity was 76% and the temperature was 64° F. At this time the child took her diet well. Her skin was not moist when she was removed briefly from the cold vapor atmosphere, and the harsh inspiratory bark had disappeared. The temperature of the child continued to remain normal, and she was removed from the "croup tent" after 18 hours and 350 cc. of 0.1% cold vapor had been used. No signs of toxicity to the medication developed and she was discharged, well, after eight days in the hospital.

TOXICITY AND SENSITIVITY STUDIES

Each patient in this series of cases was watched closely for signs of toxicity following the aerosol administration of 0.1% sodium lauryl sulfate solution. A physical examination was done daily. Rectal temperature, pulse and respirations were recorded hourly for the first 24 hours and thereafter every four hours. Frequent blood studies, including complete hemograms, bleeding, coagulation and clot retraction times were performed. The stool was examined for the presence of occult blood. Urinalyses and blood NPN were checked. One patient received 530 cc of 0.1% solution over a period of 24 hours. Another patient received but 160 cc of cold vapor over an eight hour period. No abnormal laboratory results were reported. No signs of toxic manifestations were observed in any of the patients treated in this group.

Patch test experiments with sodium lauryl sulfate were done on 122 patients to determine whether it was irritating to the skin. A 10% solution was employed in the tests. The group tested ranged in age from five weeks to 74 years. The patch tests were left on the skin after cleansing with ether, for 48 hours, and read at the end of that time and again in 72 hours. Twelve of the subjects developed a mild erythema after 48 hours. These reactions were considered to be of no significance, since 25 per cent of the patients developed a greater reaction to the adhesive used in the experiments. One patient had a mild papular reaction to the 10% dilution used. The tests demonstrated that in the concentration employed the substance employed was not a primary irritant to the skin. Because the dilution employed in the sensitivity study was one-hundred fold greater than the dilution actually used in the cold vapor, it was concluded that sodium lauryl sulfate in 0.1% concentration is probably non-irritating to the mucous membrane of the respiratory system.

Sodium lauryl sulfate (Duponol-C$^{(R)}$) has been studied by several investigators. Epstein,[5] from experiments with "irium" (sodium lauryl sulfate), concluded that the alkyl sulfates were practically free from systemic toxicity. Hall[6] studied in guinea pigs the pulmonary toxicity of wetting agents dispensed as aerosols. He noted

that these agents were toxic in proportion to their concentration. The animals were put into aerosol chambers and exposed to varying concentrations of wetting agents for eight hours a day for six days. At the end of this time the animals were sacrificed and histologic sections were made of the trachea, bronchi and lungs. None of the animals receiving 0.1 sodium lauryl sulfate exhibited any reaction or changes in the respiratory epithelium.

DISCUSSION

Patients with acute laryngotracheitis should be placed in a humid atmosphere. The disadvantage of steam for raising humidity is the high temperature which may accompany its use.

The use of nebulizers in the treatment of asthma, sinusitis and other respiratory conditions has proved effective in creating an adequate concentration of chemotherapeutic and antibiotic aerosols, to be deposited on the larynx, pharynx, trachea, upper and lower bronchial tree and in the alveoli. The size of the particle is important. The vaponefrin nebulizer used in these cases produced a droplet, 2.0 to 5.0 μ in diameter. Larger size particles are less effective in reaching all portions of the bronchial epithelium.

All the patients treated with 0.1% sodium lauryl sulfate cold vapor in our series responded dramatically. The temperature, pulse and respiration which were moderately to severely elevated on admission remissed to normal levels in less than 12 hours after beginning the cold vapor. One child was seen with a temperature of 104.2° F., pulse 204 per minute, and respirations 80 per minute and diagnosed as acute laryngotracheitis with bronchopneumonia. After eight hours in the cold vapor the temperature, pulse and respiration were normal, and he spoke to the visitors in a fine clear voice without any residual hoarseness. The rales in the left lung base disappeared in three days; the temperature remained normal and he was discharged as well after eight days in the hospital. In each new patient treated there was a repetition of the good results obtained in this patient. To date only severely ill patients had the benefit of the cold vapor. It was interesting to note that one child receiving warm steam vapor in the steam room for 48 hours had a temperature rise to 103° F. When placed in the cold vapor the temperature promptly returned to normal in four hours and did not rise again.

High fever which increases the metabolic rate and therefore the oxygen requirements, puts an added strain on the already overburdened patient. Restlessness means an air hungry patient. By quickly reducing the temperature of the patient to normal levels, the soothing cold vapor used in these patients aided in preserving their reserve

strength. The antibiotics then were more effective in combating the infectious state.

The antibiotic activity of sodium lauryl sulfate was investigated. Brain-heart infusion was used as the culture media for the hemolytic streptococcus and streptococcus viridans bacteria; 10% yeast extract was added to the media for the hemophilus influenza organism. In-vitro experiments showed that concentrations of 0.1% (1:1000) and 0.01% (1:10000) sodium lauryl sulfate completely inhibited the growth of three hemophilus influenza strains, two streptococci viridans and two hemolytic streptococci strains of bacteria. Control studies without adding the wetting agent to the infusion showed excellent growth of all the organisms.

The explanation for the bactericidal properties of sodium lauryl sulfate is not exactly clear. The cell may be rendered more penetrable by the wetting agent causing metabolic embarrassment, thereby promoting death of the bacteria.

The advantages of the simple apparatus presented in this report are multiple. It may be used in the home when hospital facilities are lacking; it costs relatively little to construct; provides humidity of 70% in a short time; a minimum of condensation without wetting the patient; oxygen concentrations above 50% at a flow of six liters per minute; an ice chamber for maintaining tent temperature between 60-65° F.; excellent visibility for the nurse and the patient. It does away with special steam rooms; discards exposure of medical attendants to water saturated rooms; eliminates water proof bedding, and the danger of scalding from steam.

Further clinical study of wetting agents in the treatment of acute laryngotracheitis and other respiratory conditions in which moisture is essential seems warranted. It is hoped that the simplicity of this inexpensive and satisfactory method for providing humidity, controlled temperature and a high concentration of oxygen in the treatment of laryngotracheitis will encourage the use of this important type of therapy in this and other conditions of the respiratory system.

CONCLUSION

1. A simple apparatus utilizing a wetting agent (0.1 sodium lauryl sulfate) for the production of cold vapor in the treatment of laryngotracheitis is presented.

2. Ten patients with acute laryngotracheitis have now responded favorably to this form of cold vapor therapy.

3. In 0.1% concentration sodium lauryl sulfate was found to be non-toxic and in 10% concentration non-allergic to the human.

4. In-vitro experiments showed 0.01% concentration of sodium lauryl sulfate completely inhibited the growth of several strains of hemophilus influenzae, hemolytic streptococci and streptococcus viridans.

(R) registered trademark.

CHAPIN HOSPITAL.

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LX

THE TREATMENT OF ENDOLYMPHATIC HYDROPS (MÉNIÈRE'S DISEASE) WITH STREPTOMYCIN

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ST. PAUL, MINN.

When streptomycin was introduced in the treatment of tuberculosis it became apparent that streptomycin had a toxic effect on the inner ear. It fell to the lot of the otologist to make observations as to the acoustic and vestibular damage during the treatment period.

At the Veteran's Hospital in Minneapolis 56 cases were placed on therapy. In 20 of these cases the therapy or tests were discontinued for various reasons. The 36 cases carried through to completion were referred weekly to the otology service for audiogram and vestibular functional tests.

During this study our attention became directed to the regularity in which abolation of the vestibular function occurred with very little, if any, acoustic damage. Since this is the result desired in the treatment of endolymphatic hydrops, we felt endolymphatic hydrops might be successfully treated by the administration of streptomycin.

The cases selected for this test were patients who stated they were totally incapacitated because of the attacks or fear of the attacks. Each case accepted treatment after a full explanation of the possibilities and probabilities. All cases were hospitalized for better observation, frequent audiograms and vestibular function tests. Only sufficient audiograms and vestibular function tests will be recorded in this paper to denote important findings and progress.

Toxic effects such as diminished renal function, eosinophilia, dermatitis, blood dyscrasia, nausea, vomiting or psychosis were not encountered in any case.

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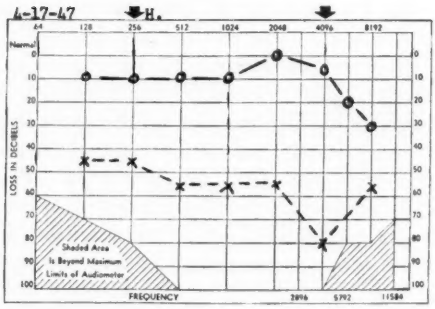
Published with approval of Chief Medical Director. The statements and conclusions published by the author are the result of his own study and do not necessarily reflect the opinion or policy of the Veteran's Administration.

Presented before the Minnesota Academy of Ophthalmology and Otolaryngology March 11, 1949.

CASE 1.—Mr. H. B., aged 38, who was first seen on April 12, 1947, gave the following history. He had had his first attacks 18 months before while on board ship. He experienced vertigo, nausea, vomiting, peculiar ringing noise and loss of hearing in the left ear. These attacks were occurring once or twice a month at their onset. For five weeks the attacks had become more frequent, occurring daily during the week preceding this first visit. Patient stated he was totally incapacitated because of attacks or fear of an attack. He had received treatment in the form of subcutaneous injections twice weekly for several weeks. He stated he was better while receiving these injections and that following discontinuance of the treatments, the attacks recurred with increased frequency. The general physical examination was negative. The neurology report showed no disease. The nose, throat, sinuses and fundi were negative. The audiogram showed a marked reduction of hearing in the left ear; the vestibular tests showed a good response from the right ear. The left ear, however, showed no response by 20 seconds cold caloric stimulations in the vertical canal. In the horizontal canal the latent period of nystagmus developed in 56 seconds and was maintained only 32 seconds. Thus we have no vertical response and a hypo function in the horizontal canal. It is not always possible to determine the exact canal involved and at times it is not possible to determine which ear is involved by the cold caloric tests. This may be due to the fact that in some of these cases we may have a bilateral involvement, a minimal on one side, and a greater amount on the opposite side, thus unbalancing our vestibular response. The reduced hearing is not sufficient to determine which is the involved side nor is the tinnitus; if the tinnitus is slight, it might be masked by the opposite side. The cold caloric stimulation test, however, appears to be the most accurate in determining which is the involved labyrinth.

The absence of vestibular function of the vertical canal and hypofunction of the horizontal canal with 20 seconds cold stimulation, the reduced hearing acuity on left, and the tinnitus on the left, all point directly to left side involvement.

On April 17, 1947, this man was placed on two grams of streptomycin daily divided into four doses. On May 3, or 16 days after treatment started, the caloric tests show no response from either the vertical or horizontal canal on the left side. Four days later the man was again tested with cold caloric stimulation 30 seconds; again there was no response from either the vertical or horizontal canal on the left side. The patient had been on treatment for 20 days and at this time it was discontinued. You will note the response in the right ear is still normal in both canals.



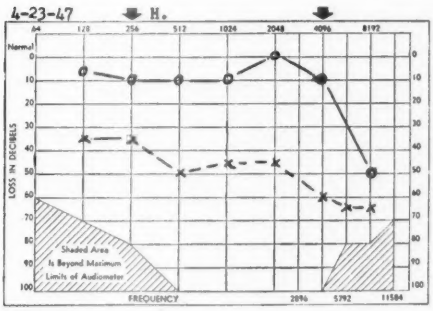
H. B. 4-17-47

Ice Water in Contact with the Drum for 20 seconds—

Right		*Left*	
Vertical Canal:		Vertical Canal:	
Developed	43 sec.	Developed	No
Maintained	53 sec.	Maintained	----
Horizontal Canal:		Horizontal Canal:	
Developed	36 sec.	Developed	56 sec.
Maintained	86 sec.	Maintained	32 sec.

This man's involved left endolymphatic system, its pathway or nuclei for some unknown reason has reacted to some unknown substance which has resulted in a group of symptoms known as endolymphatic hydrops. For the lack of a better name, let us call this unknown substance a toxin. This same vestibular apparatus, pathway or nucleus has been rendered more vulnerable to the toxin of streptomycin. Streptomycin has a selective action on the normal vestibular apparatus but a greater selective action on the vestibular apparatus in endolymphatic hydrops. The opposite, normal apparatus has not been affected by the streptomycin but would also be rendered functionless if we continued the treatment long enough. The patient received treatment for 20 days, a total of 40 grams of streptomycin. In our observation of the toxic reaction of streptomycin in patients with tuberculosis, reduced function occurred during the fourth or fifth week, these patients having received 70 to 84 grams of streptomycin. We stopped the treatment after we had rendered the affected vestibular apparatus functionless but not long enough to destroy the function of the unaffected side. This is the aim of our treatment.

In the study of hearing loss of tuberculous patients receiving streptomycin, the Streptomycin Conference[1] found 8% with a loss



H. B. 5-3-47

Ice Water in Contact with the Drum for 20 seconds—

Right		*Left*	
Vertical Canal:		Vertical Canal:	
Developed	40 sec.	Developed	No
Maintained	37 sec.	Maintained	—
Horizontal Canal:		Horizontal Canal:	
Developed	37 sec.	Developed	No
Maintained	27 sec.	Maintained	—

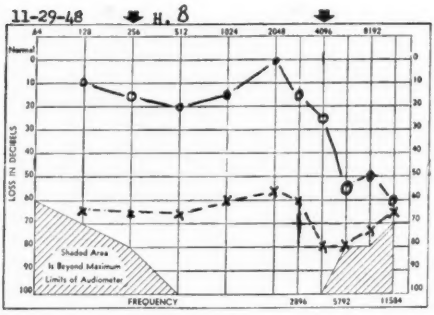
H. B. 5-7-47

Ice Water in Contact with the Drum for 30 seconds—

Right		*Left*	
Vertical Canal:		Vertical Canal:	
Developed	52 sec.	Developed	No
Maintained	76 sec.	Maintained	—
Horizontal Canal:		Horizontal Canal:	
Developed	49 sec.	Developed	No
Maintained	79 sec.	Maintained	—

of some high tones and a 4% loss of some low tones with 6% of the patients returned to normal following completion of treatment. Thus the loss of hearing is quite negligible in our therapy with the doses as given in the treatment of tuberculosis. Hearing loss does occur with extremely large doses over a prolonged period of time. Stevenson, Alvard and Carrell[2] reported autopsies on five patients who had become deaf while receiving large doses of streptomycin.

Three things happen according to the amount of streptomycin a patient receives. First, a relative small amount may produce destruction of the vestibular function in an ear affected with endolymphatic hydrops. Second, a larger amount will destroy the ves-



H. B. 11-29-48
Ice Water in Contact with the Drum

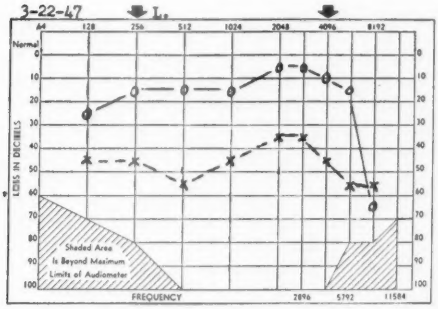
Right (20 seconds)		*Left* (3 minutes)	
Vertical Canal:		Vertical Canal:	
Developed	40 sec.	Developed	No
Maintained	55 sec.	Maintained	—
Horizontal Canal:		Horizontal Canal:	
Developed	35 sec.	Developed	No
Maintained	80 sec.	Maintained	—

tibular function of a normal vestibular apparatus. Third, cochlear damage with deafness follows a still larger daily and total dose.

This patient was recalled for examination on November 29, 1948, seventeen months and one week after discontinuing the treatment. The cold caloric test showed a good response with 20 seconds of stimulation in the right ear and no response with the 3 minutes' cold caloric stimulation in the left ear. Thus we have a normal responding vestibular apparatus in the right ear and a non-functioning vestibular apparatus in the left ear.

Two years and eleven months after completion of treatment, he had had two attacks each lasting about a minute, and several mild attacks. He does not get sick from the attacks now. Tinnitus is present to some extent, hearing acuity is the same and he has poor footing and trouble in walking in darkness. He is not incapacitated for work. His habits and food are in no way restricted because of attacks or fear of attacks.

Four years after treatment he had not had any further typical attacks. He had a few flashes of dizzy spells; they did not make him sick. Tinnitus is present in the left ear, hearing acuity is the same. Habits and food are not in any way restricted because of



L. C. 3-21-47

Ice Water in Contact with the Drum for 20 seconds—

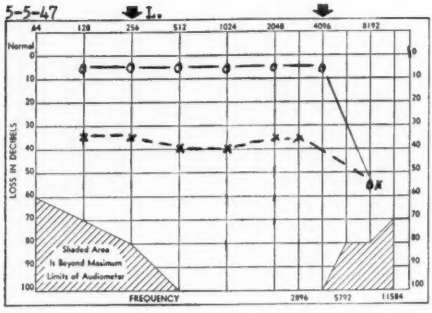
Right		*Left*	
Vertical Canal:		Vertical Canal:	
Developed	36 sec.	Developed	53 sec.
Maintained	103 sec.	Maintained	68 sec.
Horizontal Canal:		Horizontal Canal:	
Developed	33 sec.	Developed	32 sec.
Maintained	114 sec.	Maintained	123 sec.

attacks or fear of attacks and he has, at no time, been incapacitated for work.

The time element has been to this man's advantage; between the two-year-eleven-months period and the four-year period he has had a few flashes of dizzy spells but no attacks of endolymphatic hydrops.

Observation of the function of the vestibular apparatus, which had been lost during the streptomycin treatment of tuberculosis patients, showed that some of them have a return of the vestibular function. In the greater majority of cases the vestibular function is lost and may be permanently lost. In the treatment of tuberculosis with streptomycin there may be a return of function of the vestibular apparatus after it has been damaged. Also in these cases of endolymphatic hydrops there may be a return of function of vestibular apparatus with possible return of vertigo. The length of time after which one can state there will be no return of function of the vestibular apparatus with possible vertigo cannot be stated.

CASE 2.—Mr. L. C., aged 26, farmer, was first seen March 21, 1947. In June, 1945, while in service at Great Lakes he had ringing in the left ear with some hearing loss. About one month later, while



L. C. 4-11-47

Ice Water in Contact with the Drum for 20 seconds—

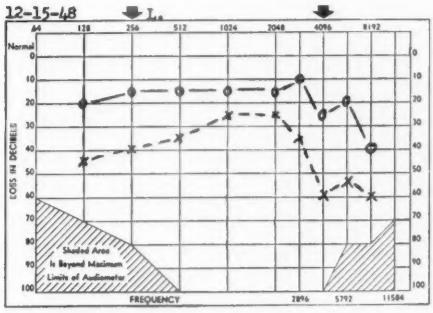
Right		*Left*	
Vertical Canal:		Vertical Canal:	
Developed	47 sec.	Developed	No
Maintained	93 sec.	Maintained	----
Horizontal Canal:		Horizontal Canal:	
Developed	30 sec.	Developed	No
Maintained	104 sec.	Maintained	----

L. C. 4-11-47

Ice Water in Contact with the Drum for three minutes—

Left	
Vertical Canal:	
Developed	No
Maintained	----
Horizontal Canal:	
Developed	No
Maintained	----

attending a motion picture, the screen started to move and blur and he had to be assisted out of the theatre. He had tinnitus, nausea and vomiting, and he staggered. He was accused of being intoxicated. The attacks become more frequent and more severe. He would be thrown to the ground by the severity of the attacks. During the previous four weeks he had had three such attacks. He stated he was totally incapacitated because of these attacks or the fear of an attack. The general physical examination proved negative. To caloric tests he had a very good response; a rather hyperactive one. With 20 second cold caloric stimulation of the right vertical canal nystagmus developed in 36 seconds and was maintained for 103 seconds. In the horizontal canal, nystagmus developed in 33 seconds and was maintained for 114 seconds. The latent period



L. C. 12-15-48

Ice Water in Contact with the Drum for 20 seconds—

Right		*Left*	
Vertical Canal:		Vertical Canal:	
Developed	No	Developed	No
Maintained	---	Maintained	---
Horizontal Canal:		Horizontal Canal:	
Developed	30 sec.	Developed	No
Maintained	95 sec.	maintained	---

L. C. 12-15-48

Ice Water in Contact with the Drum for 3 minutes—

Right		*Left*	
Vertical Canal:		Vertical Canal:	
Developed	Yes	Developed	No
Maintained	70 sec.		
		Horizontal Canal:	
		Developed	No

was 53 seconds and nystagmus was maintained for 68 seconds in the left vertical canal; the latent period was 32 seconds and nystagmus was maintained for 123 seconds in the left horizontal canal. This does not give one very much indication which canal or ear is at fault. The latent period of the left horizontal canal is prolonged. The audiogram shows the greatest reduction in the left ear and the tinnitus is also in the left ear. On March 21, 1947 he was placed on 2 grams of streptomycin daily.

By April 11, 1947 the patient had had 23 days of treatment. The caloric response to 20 seconds of stimulation was good in the right ear but there was no response in the left. Ice water in contact with the left ear drum for 3 minutes produced no response.

Again it is demonstrated that the vestibular apparatus on the affected side is more sensitive to the toxin of streptomycin than that of the normal ear. When the treatment was started the function of the right and left labyrinths was very nearly equal and after 23 days of treatment the left was completely knocked out while the right retained a normal function.

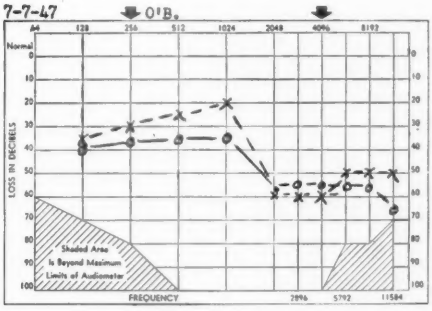
The audiogram before treatment shows that the percentage of hearing loss in the right ear is 1.6% and the left ear 38.8%. The bone conduction using the C 512 fork was 20/35 right ear and 0/35 in left. The audiogram, 23 days after treatment was discontinued, shows that the hearing was better at this time than at the time treatment was started, the percentage in the left ear being 30.6% as compared wih 38.8%.

This man was recalled for examination on December 15, 1948 or 19 months and 5 days after treatment was discontinued. He stated he had not had an attack of vertigo since leaving the hospital. He said the ringing in his ear had subsided but that he heard his heart beat in his ear. The audiogram shows a 23.4% loss of hearing in the left ear as compared with 38.8% before the treatment. The vestibular functional test showed no response in the vertical canal with 20 seconds of stimulation but a good response in the horizontal canal of the right ear. In the left ear there was no response to the 20 second stimulation. With three minute stimulation, nystagmus was maintained for 70 seconds in the right vertical canal, no response in left ear. This man is a farmer and continues his work without difficulty.

Within two years and 11 months after treatment he had had about a dozen mild attacks; there had been no vertigo. Objects moved towards or away from him and he would feel a little faint and turn pale. He had never lost his balance or fallen during the attacks. Tinnitus was present. These attacks had not interfered with full employment.

Four years after treatment he reported that he had had a total of about six mild attacks. This was in conflict with the first questionaire in which he stated he had had a dozen mild attacks. The worst attack occurred after he drank six bottles of beer. His habits and food were otherwise unrestricted. Variable tinnitus was present. His hearing had improved.

For a period of about 19 months, this man had no vertigo. Since then, however, he has had from 6 to 12 mild attacks with tinnitus. He is employed at a full-time job.



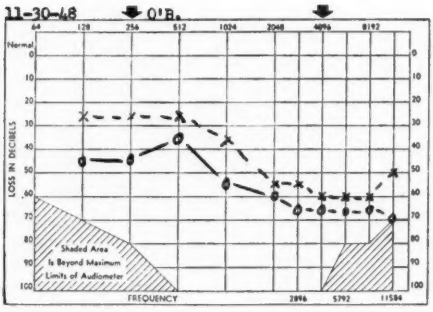
O. F. 7-7-47

Ice Water in Contact with the Drum for 20 seconds—

Right		*Left*	
Vertical Canal:		Vertical Canal:	
Developed	0 sec.	Developed	0 sec.
Maintained	0 sec.	Maintained	0 sec.
Horizontal Canal:		Horizontal Canal:	
Developed	45 sec.	Developed	24 sec.
Maintained	22 sec.	Maintained	88 sec.

CASE 3.—Mr. O. F., aged 39, electrician, was first seen in July, 1947. In 1943 while unloading cargo this man experienced a sudden attack of vertigo, became nauseated, vomited violently, had a ringing in the right ear and a sensation of extreme pressure over the frontal area. This attack lasted for two hours. Since that date he has had numerous similar attacks. During the past year the attacks have occurred two or three times a month. Since the first, the attacks have been rather mild in character, the tinnitus being most annoying. Between attacks he has difficulty weaving his way in a crowd or assuming a sudden change of position. He has noted that drinking will precipitate an attack. The audiogram shows a marked reduction in hearing in both ears.

The vestibular tests with stimulation for 20 seconds brought no response in the vertical canal of the right ear. In the horizontal canal the nystagmus developed in 45 seconds and was maintained for 22 seconds. This is subnormal reaction. There was no response to stimulation of the vertical canal of the left ear, while there was a response occurring in 24 seconds and maintained for 88 seconds in the horizontal canal. Therefore, we are dealing with a bilateral endolymphatic hydrops. The attacks of tinnitus were severe but the vertigo rather mild; this is because, in bilateral involvement, the opposing



O. F. 11-30-48

Ice Water in Contact with the Drum for 20 seconds—

Right		*Left*	
Vertical Canal:		Vertical Canal:	
Developed	No	Developed	No
Maintained	----	Maintained	----
Horizontal Canal:		Horizontal Canal:	
Developed	No	Developed	36 sec.
Maintained	----	Maintained	28 sec.

O. F. 11-30-48

Ice Water in Contact with the Drum for 3 minutes—

Right		*Left*	
Vertical Canal:		Vertical Canal:	
Developed	Yes	Developed	Yes
Maintained	32 sec.	Maintained	46 sec.

vestibular canals are not so greatly unbalanced during the attacks as they are in the unilateral involvement. Streptomycin treatment was started July 11, 1947 and discontinued 43 days later, at which time we could obtain no response with three-minute stimulation of any of the canals.

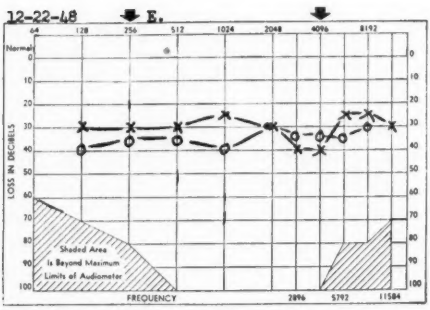
This man was retested fifteen months after the treatment was discontinued. The caloric vestibular tests with 20-second stimulation showed no response in the right ear and no response in the vertical canal of the left ear. From the left horizontal canal nystagmus developed in 36 seconds and was maintained for 28 seconds. After 3 minutes cold caloric stimulation nystagmus was maintained for 32 seconds from the stimulation of the right vertical canal and 46 seconds from the left. The function of both canals of the right ear and the vertical canal of the left ear is reduced. The left horizontal canal, however which was the normal canal at the time treat-

ment was started, still has very good function. The audiogram shows a considerable return of the lost hearing since the treatment was completed; there is a loss of 63.5% in the right ear and 46.4% in the left.

The patient stated that at the time this audiogram was made he had a head cold and he was certain that his hearing was better than was indicated by the audiogram. He also stated that he had had one dizzy spell during the preceding 15 months. He said that this was the only attack he had had and that this attack was different from the others in that he was dizzy but he did not have the sensation of objects rotating about in space. He is now fully employed as an electrician. He has a ground job, however, because of fear of climbing.

CASE 4.—Mr. E. H., aged 31, commercial painter, gave the following history when seen in December, 1948. At the age of four years he had developed suppurative otitis media with loss of hearing in the left ear. This ear drained at intervals until shortly after he entered the armed services in February, 1942. There had been no suppurative otitis media since. In June, 1944, he had an attack of nausea, vomiting, tinnitus and impaired hearing of the right ear. This attack started after the evening meal when on his way back to the barracks; he was unable to walk and had to be carried on a stretcher to the hospital. It lasted seven hours, after which he was able to return to duty. The tinnitus continued for three or four days. Two years later he had a similar attack. The next attack occurred two months later. From that time until his first visit in December, 1948, his attacks were occurring with increased frequency so that he was experiencing attacks, from one daily to one in two weeks. He stated that he was totally incapacitated because of the attacks or the fear of an attack. The general physical examination proved negative. The right canal and drum were normal. The left canal was normal. There was a large central perforation in the drum, the middle ear was dry, and no ossicles were visible. The status of his previous hearing and vestibular function was not known. Because of the perforated left drum cold alcohol was used; it was difficult to maintain a uniform temperature. Considering the suppurative otitis media in childhood with a residual perforation of the left drum without tinnitus, and because of tinnitus in the right ear with hearing loss and reduced vestibular function it was felt that the right ear only was involved.

The patient was given 2 grams of streptomycin daily for 36 days.



E. H. 12-22-48

Ice Water in Contact with the Drum for 20 seconds in the Right Ear and Cold Alcohol in the Left Ear—

Right		*Left*	
Vertical Canal:		Vertical Canal:	
Developed	No	Developed	No
Maintained	....	Maintained	....
Horizontal Canal:		Horizontal Canal:	
Developed	35 sec.	Developed	30 sec.
Maintained	50 sec.	Maintained	45 sec.

E. H. 12-22-48

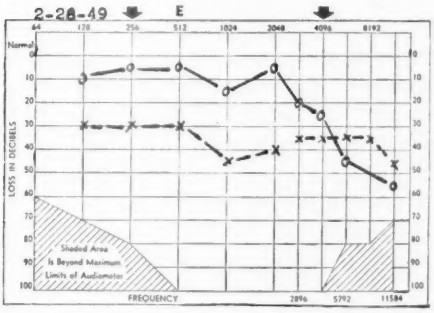
Ice Water in Contact with the Drum for Three Minutes in the Right Ear and Cold Alcohol in the Left Ear—

Right		*Left*	
Vertical Canal:		Vertical Canal:	
Developed	Yes	Developed	No
Maintained	58 sec.		

On February 28, 1949, the three minute cold caloric stimulation test showed no response in the right ear. The three minute cold alcohol stimulation of the left vertical canal produced nystagmus, maintained 60 seconds. The audiogram indicates an improvement in hearing. He has had no attacks since completion of treatment.

Within the 18 months following treatment he had had one bad attack; his hearing had remained undamaged; he had experienced tinnitus only during a cold; and his activities had not been restricted because of attacks or fear of attacks.

CASE 5.—Mr. K. G. consulted his family physician November 13, 1947, with a history of having had an attack of tinnitus, dizziness and loss of hearing in 1945. For the preceding three weeks the pa-



E. H. 1-6-49

Ice Water in Contact with the Drum for 20 seconds in the Right Ear and Cold Alcohol in the Left Ear—

Right		*Left*	
Vertical Canal:		Vertical Canal:	
Developed	No	Developed	No
Horizontal Canal:		Horizontal Canal:	
Developed	40 sec.	Developed	40 sec.
Maintained	55 sec.	Maintained	10 sec.

E. H. 1-6-49

Ice Water in Contact with the Drum for 3 Minutes in the Right Ear and Cold Alcohol in the Left Ear—

Right		*Left*	
Vertical Canal:		Vertical Canal:	
Developed	Yes	Developed	No
Maintained	90 sec.		

E. H. 2-28-49

Ice Water in Contact with the Drum for 3 Minutes in the Right Ear and Cold Alcohol in the Left Ear—

Right		*Left*	
Vertical Canal:		Vertical Canal:	
Developed	0 sec.	Developed	No
Maintained	0 sec.		
Horizontal Canal:		Horizontal Canal:	
Developed	0 sec.	Developed	Yes
Maintained	0 sec.	Maintained	60 sec.

tient had had several attacks of such severity that he was thrown to the ground and received several injuries. During February, 1948, he received a course of intravenous histamine injections. Twenty-one treatments consisting of 1 mg histamine base in 250 cc of glucose were given.

On October 25, 1949, he stated that there had been no improvement following the histamine treatments. He believed the attacks had occurred more often and were more severe. He stated he was totally incapacitated because of the attacks or fear of an attack. Beginning November 4, 1949 he was given two grams streptomycin daily for 38 days. He was free from symptoms at this time. Unfortunately the audiograms and vestibular cold caloric tests at this date are not of record. A questionnaire sent to him in April of 1950 (five months following treatment) indicates that he had had no attacks since completion of the streptomycin treatment.

After eighteen months following treatment he had had several mild attacks, the hearing was unchanged, tinnitus was the same, habits or food were not restricted because of attacks or fear of attacks. He had not been incapacitated for work.

DISCUSSION

All patients under observation had vertigo before treatment. None of them experienced vertigo immediately following completion of treatment. All experienced a varying degree of dizziness on change of position during treatment.

The rapidity with which the healthy labyrinth takes on the entire vestibular function during treatment in unilateral endolymphatic hydrops or the rapidity with which substitution occurs through the eyes, muscle sensibility, joint sensibility, deep tendon sensibility, cutaneous tactial sensations in bilateral vestibular destruction depends upon the exercise and activity which the patient maintains.

In these patients orientation was acquired in good light and firm footing; further training was necessary for darkness or soft footing. In our present day living one is not placed in darkness and soft soft footing frequently, therefore early substitution under these circumstances is not acquired. Based on the amount of streptomycin deemed necessary to produce hearing loss, it is believed that any hearing loss during, or subsequent to, the treatment is not due to the streptomycin but to the fact that the cochlea is still receiving stimulation from whatever motivates the vertiginous attacks. The effect of these attacks is not being transmitted through the now functionless vestibular apparatus, pathway, or nuclei. It is believed that still better results may be obtained by increasing the amount of streptomycin and this will be our policy in future cases in an attempt to prevent any return of vestibular function. It is believed one can increase the dose and administer streptomycin over a longer period of time and still be within the margin of safety relative to reduced function of the normal ear. It is our plan to give three grams of

streptomycin divided into two doses daily and to carry on the treatment for five days beyond the period of the loss of cold caloric stimulation response. It will be discontinued, however, immediately during this five-day period should there be any reduction of response in the uninvolved ear.

CONCLUSION

Five cases of endolymphatic hydrops have been treated with streptomycin; three cases were unilateral, one bilateral, one case an unknown quantity in one ear due to a long period of suppurative otitis media. In each case the man was totally incapacitated because of the attacks or fear of attacks. All have now been restored to full employment.

Since this report was presented six additional cases have been treated by me or members of the staff[3] with comparable results.

1596 West Minnehaha Avenue.

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Clinical Notes

LXI

NONCHROMAFFIN PARAGANGLIOMA OF THE MIDDLE EAR

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The case presented is that of a rare type of neoplasm of the middle ear, namely a nonchromaffin paraganglioma, or a glomus jugularis tumor. Lattes and Waltner[1] have termed the tumor a paraganglioma because of its apparent origin from nests of paraganglionic cells. These cell nests, similar to a carotid body, are located in the dome of the jugular bulb below the floor of the middle ear, in the canal that transmits the ramus tympanicus of the IX nerve through the middle ear floor, or in the nerve branches overlying the promontory of the cochlea.

The tumor is characterized by its slow growth, local invasiveness, vascularity, tendency to bleed, facial paralysis and aural polypoid masses presenting through the tympanic membrane and in the external auditory canal. Destruction of the temporal bone and intracranial extension may occur. Metastases have been observed in the late stages. The common subjective complaints are loss of hearing, headaches, dizziness, tinnitus and aural discharge.

REPORT OF A CASE

A 54 year old white woman was first seen Sept. 7, 1950 complaining of a pulsation in the right ear of about 12 months' duration. She had noted some diminution in hearing. Other than that there were no subjective complaints. She had had no pain, dizziness, headaches or aural discharge. There was no history of any previous ear trouble.

The right ear drum presented a red thickened appearance bulging markedly superiorly and posteriorly. It appeared to be an

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acutely inflamed bulging ear drum. The temperature was normal and there was no pain. It was not the picture of a typical acute otitis media and there were none of the accompanying general toxic signs and symptoms. The condition was treated as an acute infection with sulfonamides and aureomycin, plus intranasal medication for 13 days. The picture was the same at the end of that period. It was decided to perform a myringotomy in the office. The incision was made posteriorly through the bulging area and blood literally gushed from the middle ear. The hemorrhage was controlled after several minutes with gauze packing against the drum and in the external auditory canal. The packing was removed three days later and the drum had healed. It presented the same bulging appearance and the same pulsation was present.

The patient was admitted to the hospital and under intravenous sodium pentothal anesthesia the ear drum was again incised. Profuse bleeding was again encountered. By means of a small suction tip the field was kept clear enabling a small ear punch to be inserted through the incision in the tympanic membrane into the cavity of the middle ear. A mass was encountered and three small biopsy specimens were obtained. A preoperative diagnosis of glomus jugularis tumor was made.

At the time of the operation preparations were made to ligate the jugular vein and to expose and pack off the knee of the sigmoid sinus if uncontrollable bleeding occurred. Gas-oxygen-ether endotracheal anesthesia was used. A post-auricular incision was made and a radical mastoidectomy was performed. There was no destruction of the mastoid process by extension of the tumor. The middle ear cavity was widely exposed and the entire area was found to be filled with a reddish, purple, spongy tumor mass which bled freely. It was removed easily using an ordinary aural ring curet. There was no excessive bleeding. The tumor was removed in its entirety. The floor of the middle ear was intact and there was no apparent communication with the jugular bulb.

Microscopic section shows a growth of large polygonal cells with vesicular nuclei and eosinophilic protoplasm. These cells lie in cords separated by numerous small blood vessels lined by a single layer of endothelial cells. There are a few large cells with hyperchromatic nuclei but no mitoses are seen. These histological changes correspond to those described by Lattes and Waltner.[1]

The mastoid cavity healed rapidly. The pulsation has disappeared completely. The patient is well and complains only of the

partial loss of hearing in the operated ear, and a faint almost inaudible buzzing sound in that ear. The latter it is felt, is distinct and separate from the pulsation caused by the tumor.

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LXII

LIMITED SURGERY AFTER FAILURE OF RADIOTHERAPY IN THE TREATMENT OF CARCINOMA OF THE LARYNX

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The introduction of the Coutard principles of irradiation in the treatment of carcinoma of the larynx marked a new era in radiotherapy. The outstanding advantage of such protracted radiation is the ability to deliver a cancericidal dose to the tumor with minimal damage to the laryngeal structures and the overlying skin, in most cases.

In the earlier days of radiotherapy, the administration of concentrated, intensive radiation to the larynx in a relatively short time was frequently followed by the devitalization of the surrounding tissue and the occasional necrosis of the skin and seldom cure. However, since the employment of modern methods of protracted radiation and the increased use of several portals for therapy, few such complications should occur. In carefully treated cases, the mucositis and dermatitis, incident to radiotherapy, subsides within several weeks and the surrounding mucosa, subcutaneous tissue and skin regain sufficient vitality to tolerate considerable trauma. The blood supply may be partially compromised, yet the ability to withstand infection is not markedly diminished.

We have repeatedly had occasion to perform successfully total laryngectomy after a full course of radiotherapy had failed to cure the carcinoma. In every such instance where the tumor was found to be resectable, adequate healing and a permanent closure were obtained. In isolated cases, pedicle skin grafts had to be resorted to; but final healing was always accomplished.

The use of protracted irradiation in the hands of several observers, notably Coutard,[1] Kramer,[2] Harris,[3] Baclesse,[4] Cunning,[5]

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Read at the combined meeting of sections on otolaryngology the New York Academy of Medicine and the College of Physicians of Philadelphia on March 21, 1951.

Fig. 1, Case 2.—Microscopic section of initial biopsy showing adenocarcinoma composed of unusual mucous cells.

Lenz,[6] and Cutler[7] has often proved effective, both in eradicating the disease and in the preservation of a good voice. However, failure in some instances and an occasional severe reaction, followed by perichondritis, may be associated with this form of treatment.

The likelihood of effecting a cure and preserving the larynx in radiation therapy of carcinoma, depends on the location of the tumor and the extent of its invasion. In our experience the microscopic characteristics of laryngeal carcinoma have little or no bearing on the response of the tumor to radiotherapy. Cordal neoplasm, lesions of the ventricle and epiglottis ordinarily respond well to protracted radiation. Even after radiotherapy has failed to destroy the tumor, there still remains the probability of effecting a cure by cordectomy via laryngofissure or thyrotomy. Such limited surgery has been performed in four cases of carcinoma of the larynx not responsive to radiotherapy. Delayed, but complete healing was obtained in every instance with the preservation of an intact larynx.

REPORT OF CASES

CASE 1.—In August, 1945, N. M., a 64 year old male was referred for examination because of a three month history of hoarse-

Fig. 2, Case 2.—Low power section of the surgical specimen removed by laryngofissure. *A*, True cord. *B*, False cord. *C*, Isolated area showing minimal, residual carcinoma. Serial sections failed to show tumor elsewhere.

ness. A sessile lesion, involving the anterior third of the left vocal cord and extending up to the anterior commissure was present. Biopsy showed squamous cell carcinoma. Because of an old tuberculous infection of the right lung, surgery was not advised and a full course of radiation therapy was administered. He received 3700 x2 R measured in air, over a thirty-four day period. Except for the usual discomfort associated with radiation therapy, he did well. Six months later during routine check-up, a recurrence of the lesion on the anterior third of the left vocal cord was noted. Again, a biopsy showed squamous cell carcinoma. On February 1946, a laryngofissure was performed with removal of the left vocal cord. The post-operative course was uneventful and the wound healed promptly. He remained free of disease for the following four years and then died, with a normal larynx, of adenocarcinoma of the large bowel.

Case 2.—E. W., a 57 year old male was first seen in November, 1948, because of hoarseness. Laryngeal examination revealed an ulcerative growth on the posterior third of the right, false cord, extending into the ventricle. A biopsy was reported to show an

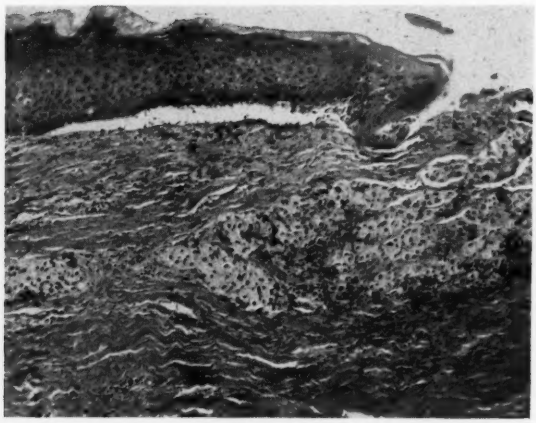

Fig. 3, Case 2.—High power section showing persistent mucous cell carcinoma, submucosally.

unusual mucous cell adenocarcinoma (Fig. 1). Laryngectomy was advised but the patient refused. He received a full course of radiotherapy, consisting of 3750 x2 R measured in air, over a thirty-three day period. Six weeks later, mirror examination showed the persistance of a small granulomatous area at the site of the original lesion. Biopsy again showed the same mucous cell adenocarcinoma. In February, 1949, a laryngofissure was performed. At operation, only minimal lesion was encountered on the right false cord, which was removed. Healing was complete but slow and accompanied by considerable slough for several weeks. Microscopic examination of the extirpated specimen (Fig. 2) in serial sections, failed to show any neoplasm except in one isolated area (Feb. 3). The patient has remained free of disease for the past two years with an intact and functioning larynx.

Case 3.—I. W., a 47 year old male first came under observation in May, 1948, because of hoarseness. Examination showed a granulomatous lesion involving the right vocal cord and extending beyond the vocal process. A biopsy showed squamous cell carcinoma. He was referred for radiotherapy and received 3900 x2 R measured in air, over a thirty-three day period. One year later, a recurrence was detected in the center of the right vocal cord and biopsy again showed squamous cell carcinoma. A laryngofissure was performed with the removal of the right true and false cords. He developed considerable post-operative edema of the right arytenoid,

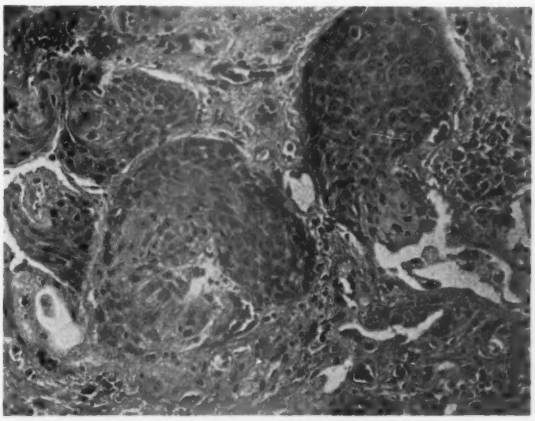

Fig. 4, Case 4.—Biopsy specimen from the granulomatous area of the epiglottis after radiotherapy, showing residual squamous cell carcinoma.

but this subsided with chemotherapy and he has remained free of disease for the past two years.

CASE 4.—O. C., a 54 year old male was first seen in June, 1950, because of hoarseness, dysphagia and occasional shortness of breath. Indirect laryngoscopy showed an extensive, ulcerative lesion of the laryngeal surface of the epiglottis. The vocal cords were not involved and no cervical nodes could be palpated. Biopsy of the lesion was reported squamous cell carcinoma. He received a full course of radiotherapy, consisting of 39 x2 R measured in air over a period of thirty-four days. The ulceration of the epiglottis improved markedly, but he developed a considerable swelling of the arytenoids and aryepiglottic folds. Three months later, there was still a small granulomatous area on the distal third of the laryngeal surface of the epiglottis. Biopsy from this region failed to show any neoplasm. His speech improved, but because of the persistance of the ulceration, another biopsy was done. This time fragments of squamous cell carcinoma were detected (Fig. 4). After preliminary tracheotomy a trans-hyoid pharyngotomy was performed and the enlarged edematous epiglottis was delivered (Fig. 5). The small, ulcerative area noted on mirror examination was found to be completely surrounded by an intact, normal mucosa (Fig. 6). No other evidence of tumor could be seen anywhere in the larynx. The epiglottis was excised. A frozen section of the edges showed the line of incision to be wide of the growth. Recovery was slow and ac-

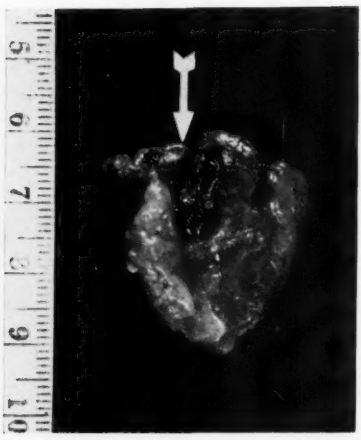

Fig. 5, Case 4.—Gross picture of the edematous epiglottis removed by transhyoid pharyngotomy. The ulcerated area on the laryngeal surface of the epiglottis, near its free edge, can be seen.

companied by considerable edema and slough at the mucosal suture line. Microscopic examination of the epiglottis, in serial sections, showed only the smallest residue of tumor at the site of the ulceration. In fact, it was difficult for the pathologist to locate the neoplasm. After four months, healing was complete and the patient was decannulated. He is continuing to do very well and has returned to his normal occupation.

These four cases are of interest because they tend to dispel the current notion that limited surgery cannot be safely performed after a full course of protracted, carefully administered radiation. In these instances, the post-operative course was uneventful, although healing was somewhat delayed.

Another provocative phase of these cases is the effect of radiotherapy on the neoplasm, in cases in which cure was not attained by radiation. In Cases 2 and 3, a carcinoma which was initially not suitable for laryngofissure, was rendered amenable to such limited procedure. When thyrotomy was contemplated in these cases, the patients were advised of the possible need for more extensive surgery and permission for laryngectomy was obtained. A frozen section of the removed specimen gave the final assurance that the excision was wide of the neoplasm.

Cases 2 and 4 illustrated another, most interesting result of radiotherapy on the tumor. Although the entire lesion was not sterilized, the major portion was destroyed, leaving only a minute lesion. It may well be that this small residue represents the original parent cell, the daughter cells having been destroyed. It would seem that the idea that a neoplasm is either entirely radiosensitive or radioresistant, is fallacious. Certainly, in these two cases, the residual carcinoma was barely detected by the pathologist, after biopsy had been done.

Fig. 6, Case 4.—Saggital section of the epiglottis showing the ulcerative tumor-containing area which is completely surrounded by uninvolved edematous mucosa. *A*, Laryngeal mucosa surface appears completely intact except at the site of the tumor. *B*, The laryngeal surface of the epiglottis. *C*, The fragmented epiglottic cartilage which probably resulted from the fibrous invasion associated with radiation.

Every so often, in the treatment of carcinoma, the larynx is rendered free of disease either by thyrotomy or radiotherapy only to develop cervical metastases. Most probably, such local lymph node invasion is the result of lymphatic emboli from the original tumor. It appears possible, in such instances, to perform a radical neck dissection for cervical metastases without disturbing the intact larynx.

CASE 5.—H. W., a 55 year old male first came under observation in October, 1946. He had an infiltrating lesion of the upper surface of the left vocal cord, extending into the ventricle. A biopsy report was squamous cell carcinoma. A laryngofissure was performed

with the removal of both the left true and false cords. Five months later, in March, 1947, a firm node was detected in the left posterior, cervical triangle. The larynx was completely healed and showed no evidence of disease. The node was excised and proved to be metastatic squamous cell carcinoma. The patient received a full course of radiotherapy to the left neck. For the following three years, he remained symptom free without any evidence of recurrence. In March, 1950, a firm gland could be palpated under the left sternomastoid muscle. This was aspirated and found to be carcinomatous. A left radical neck dissection was performed one year ago and he is at present free of disease, with an intact larynx, four and one half years after the original thyrotomy.

CASE 6.—A. R., a 37 year old male was first seen in June, 1947, with a squamous cell carcinoma of the right false cord and ventricle. He was treated by radiotherapy and received 3800 x2 R measured in air, over a period of thirty-seven days. The lesion disappeared and the larynx has remained uninvolved. Three years after radiotherapy, he developed a mass of firm nodes under the left sternomastoid muscle. A left radical neck dissection was performed and he has remained free of disease ever since, with an intact larynx.

These two cases demonstrated the feasibility of doing radical neck dissection, without disturbing a larynx which had been rendered free of disease, either by thyrotomy or radiotherapy.

SUMMARY

Modern methods of protracted radiotherapy for carcinoma of the larynx permit the administration of a cancericidal dose of radiation to the neoplasm, without severe devitalization of the surrounding structure and skin in a considerable percentage of patients.

Delayed but ultimate, complete healing can be expected after laryngofissure or thyrotomy in cases previously adequately treated by radiotherapy. The use of antibiotics and chemotherapy does much to enhance the healing properties of the irradiated tissue. Conservative surgery may be employed in such instances to eradicate residual disease in selected cases of limited carcinoma of the larynx. Thus, the original intention of affecting a cure and preserving laryngeal function may still be realized, in spite of previous, unsuccessful irradiation.

It is feasible to perform radical neck surgery for metastatic carcinoma without disturbing an intact larynx which has been previously rendered free of disease, either by thyrotomy or radiotherapy

The ultimate outcome as regards control of the laryngeal disease will have to be determined after a more prolonged follow-up. The preservation of laryngeal function in these reported cases has been encouraging.

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LXIII

AN UNUSUAL CASE OF MULTIPLE FOREIGN BODIES

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The following case is reported; not because of any unusual difficulties encountered in removing the foreign bodies, but because of the pathological condition produced by their long sojourn in the throat and the important part the antibiotics played in the patient's recovery.

Our patient, a man of 74 years, was admitted to the medical service of Fordham Hospital in a state of coma. He had been found lying on the floor of his room unconscious. He was emaciated and dehydrated, and had a temperature of 103°, a pulse of 140; the blood pressure was: systolic 139, diastolic 91.

There was a fetid odor to the breath and considerable enlargement of the anterior and posterior cervical glands.

Examination disclosed a dry crusted mucosa of lips, cheeks, tongue and pharynx. All the teeth had been extracted. On mirror examination all the structures of the larynx were found to be bathed in a foul muco-purulent material which overflowed into the trachea. In this well of pus, two grayish objects were seen wedged in both pyriform fossae. The mucous membrane throughout was red and edematous; in the region of the pyriform fossae it was black and gangrenous.

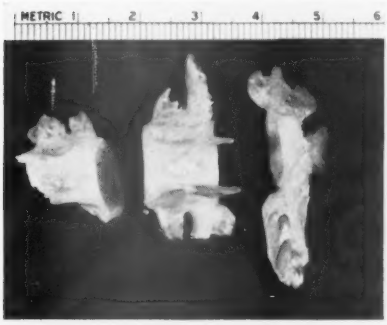
X-ray studies were reported as follows:

(1) Multiple foreign bodies (bones) present above and below the level of the cricopharyngeus muscle.

(2) No evidence of foreign body in the lungs, but some consolidation and infiltration of both bases.

One thousand cc of 5% glucose in saline solution was given intravenously and 600,000 units of penicillin was given (intramuscularly).

Morphine sulphate, grain 1/8, and atrophine sulphate, grain 1/150, was the only preparation needed for endoscopy.



Three bones were removed without incident. One was lodged in each pyriform fossa and the third below the level of the cricopharyngeus.

The mucous membranes in these areas appeared swollen, necrotic and gangrenous and bled easily. It was thought that the presence of a feeding tube at this time might cause a perforation, if indeed one was not already present.

The post-operative regimen was as follows: nothing by mouth; 1000 cc 5% glucose in saline intravenously q. 8 hrs.; Boraca C. 1 ampule daily; Amigen 5% in 5% dextrose. 1000 cc intravenously q. 24 hrs.; 300,000 units penicillin intra-muscularly, q. 12 hrs.; liver extract, 1 ampule daily; 300 cc of whole blood.

An effort was made to keep the mouth and throat clean by gentle aspiration with a soft rubber catheter; the patient's position in bed was changed frequently, the head slightly elevated.

Indirect examinations were made every few days and on the seventh day a dirty gray slough had separated from the pyriform fossae, a great deal of the edema had disappeared, and there was no longer an overflow of secretion into the trachea.

At this time a feeding tube was passed; the glucose was given intravenously only once daily for another three days. A well balanced diet was given through the feeding tube, which was allowed to remain in place for one week. Upon its removal the patient was able to swallow liquids and semi-soft foods without difficulty.

300,000 units of penicillin were given every 12 hours for fourteen days and then continued once daily for another week.

The vitamins, amino acid and liver extract, were given for a total period of three weeks.

The culture of pus taken at the time of removal of the foreign bodies showed a mixed infection of staphylococcus aureus and hemolitic streptococcus.

During this time the usual laboratory studies were carried out, including blood counts, blood chemistry, and urinalysis.

The patient's temperature gradually subsided, returning to normal on the eighth day. He showed signs of returning consciousness on the fourth day and was able to recognize those about him on the seventh day. From then on his condition improved rapidly.

We were naturally interested to know how he managed to swallow the second and third bones, when the first one must surely have given him quite a jolt. Before his transfer to a convalescent home, he confided in us that prior to going out to dinner that night he had imbibed too freely. The fish dinner was especially appetizing, and a new set of false teeth did the rest. He remembered going back to his room and having a sore throat and a bad cough the next day. From then on the picture was vague. It was established that five days had elapsed before he was brought to the hospital.

2615 GRAND CONCOURSE.

The Scientific Papers of the American Broncho-Esophagological Association

LXIV

CONGENITAL ANOMALIES OF THE ESOPHAGUS

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AND

WILLIS J. POTTS, M.D. (By Invitation)

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A review of the subject of congenital anomalies of the esophagus is presented because of the apparent frequency of these anomalies and because of the increasing significance as an endoscopic problem both pre- and post-operatively. While the surgical aspects of the lesions will be mentioned herein, the technics of surgery will not be discussed since detailed surgical descriptions of operations used in this series have been previously reported by one of us (W. J. P.) elsewhere.[1]

Classification: Congenital anomalies of the esophagus are divided into three categories: 1. Those involving the esophagus alone, 2. those in which the trachea is associated in the anomaly, and 3. those in which anomalies of other structures influence the anatomical configuration and thus the function of the esophagus. Such a classification is as follows:

I. Anomalies of the esophagus alone
    1. Absence
    2. Duplication
    3. Atresia
    4. Webs
    5. Stenosis
        a. Fibrous strictures
        b. Failure of epithelization

6. Short esophagus
7. Achalasia (cardiospasm)
    a. Simple achalasia
    b. Extreme dilatation with fibrous stricture at cardia

II. Combined esophago-tracheal anomalies
    1. Tracheo-esophageal fistula with esophageal atresia.
    2. Tracheo-esophageal fistula without esophageal atresia

III. Esophageal anomalies due to congenital anomalies of other structures
    1. Obstructions due to cardiovascular anomalies
    2. Lung buds of esophageal origin

I. ANOMALIES OF THE ESOPHAGUS ALONE

1. *Absence:* Complete absence of the esophagus has not been encountered in our series. It is described by Terracol[2] as extremely rare; the hypopharynx terminates in a cul-de-sac and the cardia is completely sealed below the diaphragm.

2. *Duplication:* Langston, Tuttle and Patton[3] have recently reviewed the subject of esophageal duplications. They present two cases, one of which had a duplicate esophagus tubular in form, indistinguishable grossly from the true esophagus, extending from the neck to well below the diaphragm. It paralleled the esophagus and passed into the abdomen through a hiatus of its own.

One case of duplication of the foregut is reported in this series. It is similar to those reported by Dickson, Clagett and McDonald,[4] Baar and D'Abreu,[5] and others in that while it was found to lie in the thorax and was connected to the esophagus by a strand of fibrous tissue, it contained considerable gastric mucosa with typical peptic ulcers. The infant was 21 months of age at the time of hospitalization and the symptoms were those of cough, intermittent fever, occasional dyspnea and failure to gain weight. Roentgenograms revealed a cyst in the right chest. Since at one time the cyst was entirely homogeneous, bronchoscopic and bronchographic studies were made in an attempt to demonstrate a bronchus leading to the cyst. However, the bronchogram showed only compression of the lung. The cyst was unrelated to the bronchial tree and it was removed surgically (W. J. P.). It was considered to be a duplication of the esophagus containing gastric mucosa lying in the central portion of the chest. It was apparently free from the esophagus although a

retropleural tube-like structure 3 mm in diameter was found leading upward from the cyst into the mediastinum.

3. *Atresia:* Complete atresia of the esophagus without an associated tracheo-esophageal defect is relatively rare. It has been encountered three times in our series. Haight[6] reported three similar cases in 1946. This form occurs in numerous variations with the two segments varying in length downward from the pharynx and upward from the diaphragm. In some cases a strand of fibrous tissue joins the two segments although generally they are entirely separate.

The symptoms of complete esophageal atresia without a tracheo-esophageal fistula are usually indistinguishable from those of the infant who has an associated fistula. The infant coughs, chokes and turns cyanotic with feeding attempts and immediately regurgitates the first and second swallow. Saliva is found drooling from the dependent corner of the mouth as the infant lies in the bassinette. On x-ray, however, no air is found under the diaphragm in the stomach or bowel although 0.5 cc of Lipiodol given by mouth shows the typical meniscus characteristic of esophageal atresia (Fig. 1). Another finding is the absence of fetal hair in the meconium stools, a constant finding in the stools of normal new-born infants, as well as in infants with esophageal atresia and a tracheo-esophageal fistula.

From a theoretical standpoint, the infant with a complete esophageal atresia without a tracheo-esophageal fistula should present a favorable surgical prognosis because of the absence of regurgitated gastric content in the tracheobronchial tree. The chemical pneumonia which complicates the aspiration pneumonia in the infant with a fistula adds an additional hazard which is significant. However, this is more than offset by the fact that the two ends of the esophagus are so widely separated in the complete atresia without a fistula, that direct anastomosis without immobilization of the stomach is impossible and the surgeon must resort to alternatives such as gastrostomy and external pharyngotomy as the primary procedure, with reconstruction of the pharyngo-gastric continuity at a later date. One such case successfully operated is reported as follows: M. H., aged 2 days, weighing 5 pounds and 3 ounces, was brought to the Children's Memorial Hospital in an oxygen cabinet, December 12, 1947. At birth much fluid was aspirated from the throat before normal respiration was established. Thick mucus constantly collected about the baby's mouth. Injection of 2 cc of Lipiodol through a catheter into the upper end of the esophagus revealed atresia. Absence of air in the gastrointestinal tract indicated atresia without communication with the trachea. The lungs were relatively

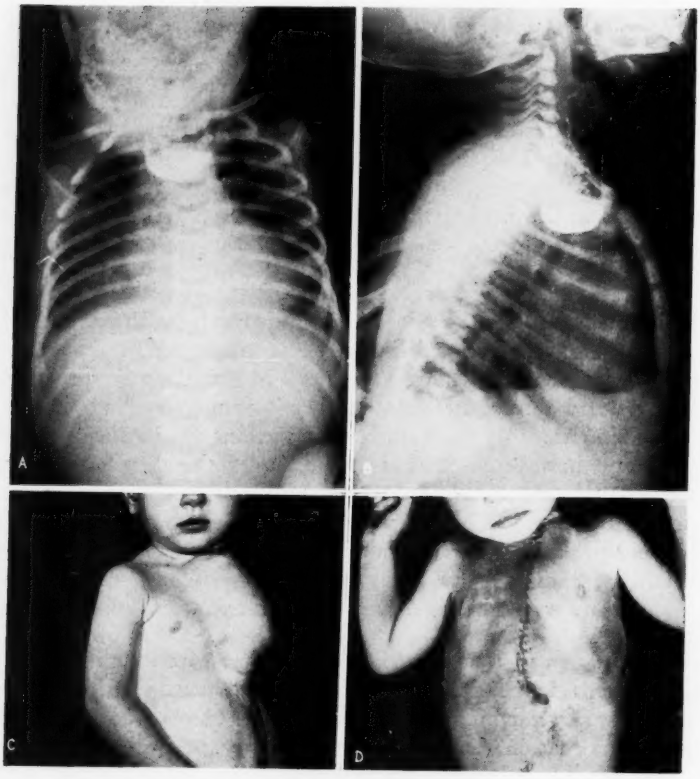

Fig. 1.—*A* and *B*. Anteroposterior and lateral x-rays of infant with congenital esophageal atresia without tracheo-esophageal fistula. Note absence of air below the diaphragm. *C*. Infant at 1½ years of age with large, bulging, subcutaneous stomach. *D*. Suture line after section of clavicle and all ribs on left side to permit placement of stomach in thorax.

clear. General physical examination revealed no other deformities. On December 13 an exploratory operation revealed that the segments of the esophagus were separated by almost three inches and could not be brought together by traction; a gastrostomy was done two days later. Because transplantation of the stomach into the chest with anastomosis of the fundus to the upper end of the esophagus inside the chest was contemplated, exteriorization of the upper end of the esophagus at the neck was delayed. Almost constant aspiration of pharyngeal mucus was required. Endoscopic aspiration of bronchial secretions was done on numerous occasions.

On January 16, 1948, the upper end of the esophagus was exteriorized above the left clavicle and the infant was discharged from the hospital February 4. On September 28, 1948, at the age of 10 months the child, now weighing 15 pounds and 6 ounces, was readmitted to the hospital and an anastomosis between the cervical end of the esophagus and the fundus of the stomach was accomplished by freeing the stomach from the abdominal wall and carrying it upward beneath the skin to the end of the cervical esophagus. The first few months after operation the subcutaneous stomach was not excessively large, but thereafter it increased markedly in size although it functioned perfectly. Because of the increasing size of the stomach, the child, now 22 months old, was readmitted to the hospital and the following operation was performed (Fig. 1-*C, D*). A long skin incision was made from the left clavicle to a point midway between the xiphoid and the umbilicus. The stomach was easily separated from the skin and sternum. Beginning at the lower margin, the costal cartilages from the seventh to the second inclusive were cut slightly lateral to the sternocostal junction. As the upper ribs were cut the left pleural cavity was opened. The medial portion of the clavicle and a similar segment of the first rib were removed with the periosteum.

The diaphragm was split straight back to the vertebrae and the stomach was placed in the left pleural cavity behind the lung and the heart, and fixed to the pleura in this position. The diaphragm was repaired allowing sufficient room for stomach emptying. Convalescence was stormy, but the child gradually began to take fluids and food and on the fourteenth day after operation was discharged in very good condition.

During the first month after operation she gained a pound in weight. Now 18 months after the final operation, she is doing very well, eats four meals a day and plays vigorously. Respirations were somewhat rapid for a time but are now normal.

4. *Web:* Single or multiple webs of the esophagus are not common. They are differentiated from congenital esophageal stenoses by their roentgen and esophagoscopic appearance only since the symptomatology is the same as that of esophageal stenosis. Dysphagia and regurgitation and a history of slow feeding habits and that the child is a "feeding problem" are usually elicited. The x-rays show the lumen of the esophagus to be of normal caliber with the exception of single or multiple shelves of mucosa indenting the lumen in thin web formation (Fig. 2). No rigidity of the esophageal walls is noted as is seen in congenital esophageal stenosis where rigid fibrosis is seen adjacent to the stenotic area. Esophagoscopically the web is

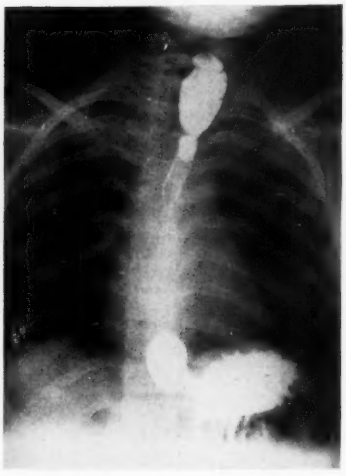

Fig. 2.—Congenital esophageal web which had caused dysphagia since birth. Response to esophagoscopic dilatation was prompt.

seen as a thin mucosal obstruction of the lumen that is soft and responds easily to the passage of bougies and the esophagoscope through its lumen.

5. *Stenosis:* Two distinct types of esophageal stenosis have been encountered in our series of congenital esophageal anomalies. One is the tight fibrous stricture involving segments of the esophagus which vary in length from one to 10 cm (Fig. 3). The other appears to be a failure of epithelization of a segment of the esophagus (Fig. 4). If the obstruction in either of these types is severe, dysphagia, regurgitation, slow feeding time, loss of weight or failure to gain weight will be apparent in the first few days or weeks of life. However, infants with these anomalies usually present no symptoms until they are about five months of age.[7] During this time they are on a completely liquid diet and fluids pass through the stenotic areas without appreciable hesitation. When semi-solid foods are added to the diet, feeding problems manifest themselves by slow eating and a reluctance to take anything but liquids. Often the family becomes accustomed to the length of time it takes to feed such an infant, but the child is sooner or later labeled a "feeding problem." When solids are added to the diet, frequent episodes of temporary obstruction occur with drooling, vomiting of undigested food and loss of weight. The child soon realizes he must refuse solid food entirely, thus fur-

ther leading to the supposition that he is a "feeding problem." Bizarre eating habits develop, with the result that as the child grows older the time used to consume a meal may extend to an hour or more. Finally, episodes of complete obstruction occur which require esophgoscopic removal of a foreign body—a piece of meat, an orange seed, a toy or a coin.

Thus, the history of a congenital stenosis of the esophagus is quite characteristic of that of obstruction but the history gives no information as to the type of anomaly which is present. Differentiation between a congenitally short esophagus and the congenital esophageal stenoses is best made by roentgenographic and esophagoscopic examinations. The roentgenograms of a child with a congenital stenosis demonstrate a prominent dilatation of the upper portion of the esophagus, with a thin, irregular strand of barium sulfate of varying length from the point of the stenosis to the diaphragm and to the stomach itself. Such a stenosis may be 1 to 10 cm in length, depending on the age of the child; if the area of stenosis is short it suggests the appearance of a cardiospasm. Esophagoscopically, such areas of stenosis are found abruptly terminating what appears to be a normal esophageal lumen. They lie in the middle of the thorax halfway to the diaphragm or below this point. The mucosa immediately around the stenosis is severely inflamed and often covered with granulation tissue, and the stricture sometimes responds readily to dilation, bougies passing into the stomach without appreciable resistance. After gradual dilation with bougies, an esophagoscope may be advanced through the stricture to continue the dilatations. In some cases response to dilatation of the congenital fibrous stenosis of the esophagus is extremely slow and difficult, and occasionally it is impossible to establish a lumen through the strictured area by endoscopic means. Gastrostomy and retrograde dilatation have been necessary in several of our cases, while in one case a surgical resection of the stricture was necessary.

Of considerable interest have been the soft stenoses of the distal half of the esophagus which appear to be due to a failure of epithelization of the lower half of the esophageal lumen. The history of dysphagia and, in older infants and children, of foreign body accidents is as described above. X-rays demonstrate a normal or slightly dilated upper half of the esophagus with a thin hair-like line of the opaque medium descending from this point to the stomach. Esophagoscopy shows the upper esophagus to be essentially normal except for the retention of fluid. The normal esophageal lumen terminates abruptly in the mid-esophagus and a small, red lumen is visable descending from the center of the stenotic ring. The esoph-

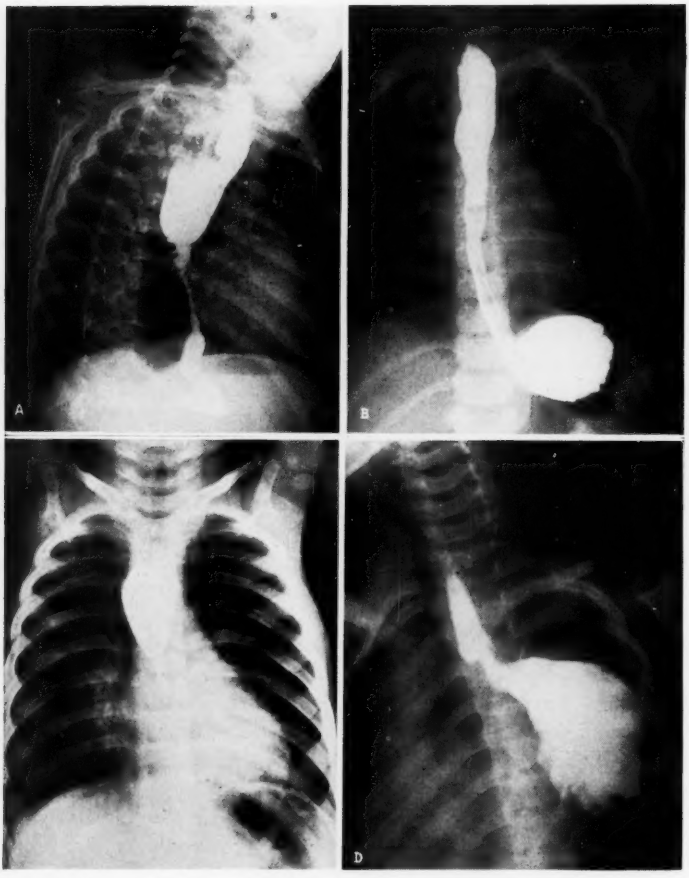

Fig. 3.—Two infants with congenital esophageal stenosis. *A.* Original x-ray showing marked stenosis of distal half of esophagus. *B.* Response to esophagoscopic dilatation. *C.* Second infant with similar area of stenosis which did not respond to dilatations, necessitating surgical correction. *D.* Post-surgical x-ray following resection of stenosis and anastomosis of stomach to upper esophagus.

agoscope may generally be readily advanced into the soft, granular inflamed lumen below which does not have the normal esophageal mucous membrane covering. This area remains inflamed until subsequent dilatations eventually relieve the stenosis and the granulation tissue is replaced by a smooth although considerably reddened mucosal surface. These strictures respond rapidly to dilatation and often following the first or second dilatation the child is able to eat normally and his entire personality changes. This lesion is not to be confused with the congenitally short esophagus since biopsies of the esophageal wall taken from below the stricture show only granulation tissue and no gastric mucosa, and x-rays demonstrate the normal stomach outline entirely below the diaphragm.

6. *Short esophagus:* The so-called congenitally short esophagus presents an entirely different picture both roentgenologically and esophagoscopically from those of esophageal stenosis described above. The following description of the roentgenologic findings by Clerf and Manges[8] is significant: The essential points in the roentgen ray diagnosis of congenital shortening of the esophagus are, first, a portion of the cardiac end of the stomach must be shown to stay above the level of the diaphragm; second, the esophagus must be shown to be too short to reach as low as the level of the diaphragm. As to the first point, the only characteristic sign is the presence of longitudinal rugae markings in that portion of the tube just above the diaphragm (Fig. 5, *D*). When this point is established, then change of posture will not cause that portion of the stomach above the diaphragm to go either lower or higher, and there will be no variation in relations on repeated studies.

Cases of congenital shortening of the esophagus are almost invariably associated with a point of pronounced narrowing at the true cardia. It is at this point that the foreign body becomes lodged, and the condition is first recognized after the removal of the foreign body and confirmation by roentgenologic studies. The area of narrowing is generally at the level of the seventh or eighth dorsal vertebrae.

Esophagoscopically, the congenitally short esophagus has an entirely different appearance from that of the congenital esophageal stenosis. The mucosa at the cardia which now lies well above the level of the diaphragm is smooth and the stricture is firm. An esophagoscope can be passed through the stenosis only with great difficulty, and response to dilation is not nearly as satisfactory as the response to dilation of the congenital esophageal stenosis. If the esophagoscope can be passed through the stenotic area, gastric rugae are visualized immediately below the stenosis with the characteristic bright

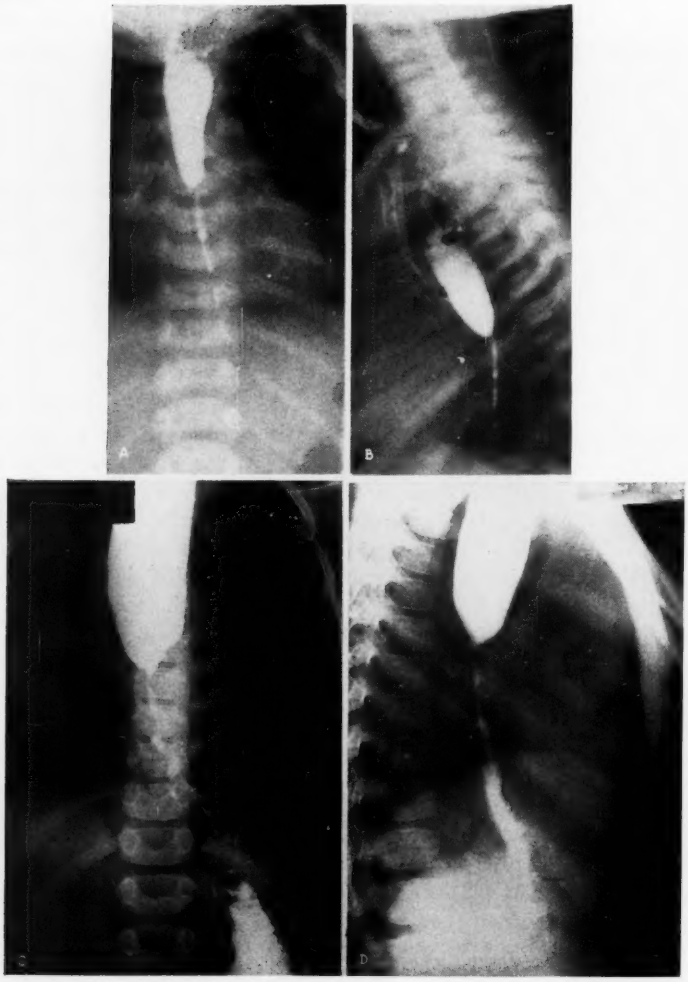

Fig. 4.—Congenital esophageal stricture apparently due to failure of epithelization of lower half of esophagus. *A* and *B*. Infant 3 weeks of age. *C* and *D*. Similar picture in 5 year boy with history of dysphagia since birth.

red, velvety appearance of gastric mucous membrane. Treatment again is repeated dilation by any of the methods commonly employed in benign esophageal strictures. The response to treatment is not as satisfactory as for most cases of congenital stenosis.

7. *Achalasia* (*cardiospasm*): Cardiospasm is one of the least understood conditions of the gastro-intestinal tract. Conclusive evidence of this becomes apparent when one reviews the literature dealing with this disease and finds it described by almost unending list of terms. A few of them are: preventriculosis, achalasia or ectasia of the esophagus, phrenospasm, mega-esophagus, megalo-esophagus, hiatal esophagismus, idiopathic or diffuse dilatation of the esophagus. Briefly stated, cardiospasm is the sydrome of non-organic obstruction of the cardia, associated with marked dilatation and hypertrophy of the esophagus (Fig. 5). It is generally considered to be a failure of coordination of the mechanism at the cardia which prevents normal passage of food from the esophagus to the stomach. In some cases, however, a tight musculo-fibrous stricture is found at the cardia similar to that seen in pylorospasm.

A review of the literature[9] and an analysis of the three cases of cardiospasm in new-born infants previously reported,[10] and three additional cases seen since that report, demonstrate that a high degree of so-called "cardiospasm" can be present at birth. It has shown, too, that the condition can develop in the first few months or years of life to give a picture identical to that seen in the adult. In these infants no vitamin deficiency was demonstrated and, in some, following the single dilatation no return of symptoms occurred although there was no change in diet.

The treatment of cases of cardiospasm in infants varies from the simple endoscopic dilatation of the cardia to the surgical esophagogastric anastomosis. In several cases previously reported, the dilatation of the cardia done with a relatively small esophagoscope, 6 mm, completely cured the condition. In other instances, however, a gastrostomy and retrograde dilatations had to be done, while in still others even dilatations with the esophagoscope or retrograde dilators failed to give permanent relief. In one infant with cardiospasm associated with stricture formation a Ramstedt type of operation was employed after dilation was found to be unsuccessful.

Aside from the mechanical or surgical treatment, other factors important in treatment should be stressed. Atropine or benzedrine in small doses is of definite assistance to some patients during times of particular stress. The administration of adequate doses of vitamin B_1 seems indicated, although there is no agreement on the ef-

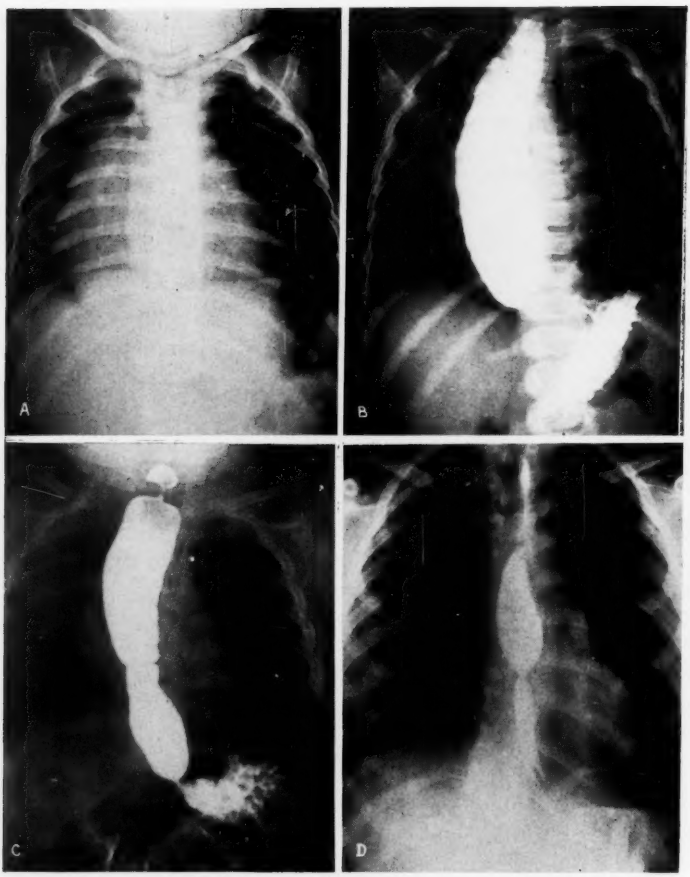

Fig. 5.—Cardiospasm and congenitally short esophagus. *A.* Infant with symptoms of esophageal obstruction and dyspnea since birth. The apparently widened cardiac shadow is due to the dilated fluid containing esophagus. *B.* The same infant following ingestion of barium. *C.* Esophagram of a new-born infant with dysphagia showing typical "cardiospasm." *D.* Congenitally short esophagus in contrast to *A*, *B* and *C*. Gastric mucosa is present below the stricture at the cardia.

ficacy of this treatment. Since degenerative changes in Auerbach's plexus are not reversible the administration of thiamin chloride could only theoretically prevent a progression of the disease rather than cure it.

The management of diet in the new-born is not a problem except that food is repeatedly regurgitated. Warm fluids or soft foods are tolerated best by all patients, whether adults or infants. Peculiar is the fact that most infants and children with cardiospasm gain rather well in spite of vomiting large portions of each meal. In late cases, however, loss of weight and actual starvation become apparent.

Of considerable concern have been the pulmonary complications of cardiospasm as well as of some of the other types of esophageal stenosis. Extensive bronchiectasis in one boy which resulted from aspirated esophageal contents prevented surgical repair of a far-advanced cardiospasm. This complication is a late one, but serious enough to contraindicate surgery when it has become fully developed, and thus must be considered in all early cardiospasms that fail to respond to dilatation. If it is considered as a possible sequela of failure of conservative measures of treatment, the surgical repair will in all probability be recommended before the cardiospasm becomes too extreme. Postural drainage should be carefully practiced by patients in whom coughing and choking are a part of the symptom complex, especially if occurring during the night.

II. COMBINED ESOPHAGO-TRACHEAL ANOMALIES

1. *Tracheo-esophageal fistula with esophageal atresia:* The principal types of congenital esophageal atresia with tracheo-esophageal fistula are well understood.[11, 12, 13] Commonest, 93% of our cases, consists of an upper blind pouch ending at or slightly above the level of the fourth thoracic vertebra. Its lower segment from the stomach is connected to the trachea, carina or left main bronchus by a tract of varying diameter (Fig. 6). In the second type, the upper segment enters the trachea while the lower segment is blind, and in a third variation of this anomaly both segments enter the trachea and may or may not be connected to each other.

The symptoms of infants with congenital esophageal atresia are classic. As the new-born infant is fed, the first two or three swallows are normal, then suddenly the fluids return through the nose and mouth; the child coughs, struggles, turns cyanotic, and may stop breathing. The cycle is repeated with each attempt at nursing, and between feedings there is a constant flow of saliva from the dependent corner of the mouth. In the commonest type the stomach be-

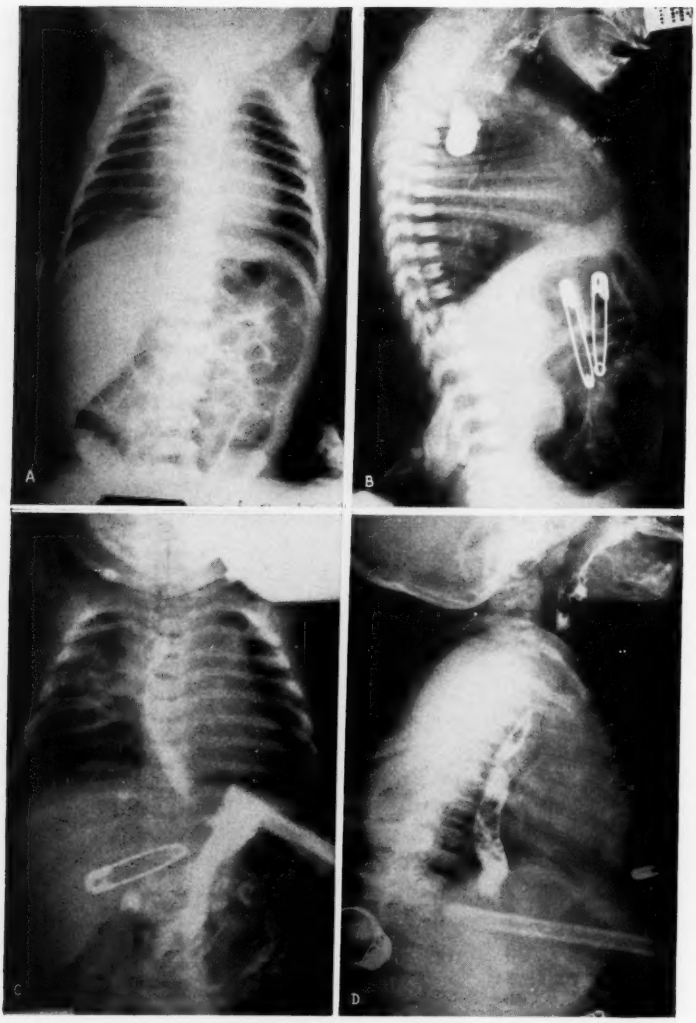

Fig. 6.—Congenital esophageal atresia with upper blind pouch and lower segment attached to the trachea. *A.* and *B.* demonstrate the preoperative appearance with 1 cc of Lipiodol. *C* and *D.* show a post-surgical stricture which has required retrograde dilatations. There was no associated tracheal stenosis in this infant.

comes distended with air, and bile and gastric secretion may be collected from the regurgitated material because of the fistula between the lower segment and the trachea. The diagnosis of congenital atresia of the esophagus is made by the history of difficulty in swallowing, choking, cyanosis, or regurgitation with the first or second swallow of food, and may be confirmed by roentgenologic catheter, or esophagoscopic evidence of atresia. Iodized oil rather than barium should be used at the contrast medium for roentgenologic studies to avoid the danger of aspiration of barium.

In the past, treatment of these atresias has been of little avail. Death was usually due to bronchopneumonia and starvation. Gastrostomy resulted in flooding the lungs with gastric content after feeding unless the cardia was ligated at the time of the gastrostomy. A slight improvement in results was obtained following gastrostomy and ligation of the cardia when an esophageal fistula was established in the neck bringing the upper esophageal segment to the outside to permit external drainage of the pharyngeal secretions to avoid aspiration into the tracheobronchial tree. Several children are now living who have had this type of surgical treatment, followed by an anastomosis by a skin tube flap between the esophageal fistula in the neck and the gastrostomy opening. During the past four years, 46 infants with congenital atresia of the esophagus have been seen at the Children's Memorial Hospital.[14] Of these, 43 were found to have the type of atresia in which the upper segment is blind and the lower segment is connected to the trachea. In the remaining three cases both segments were blind; one of these, reported above, is living and well.

Recent advances in surgical and anesthetic technics have made it possible to perform direct anastomosis between the upper and lower segments of the esophagus principally in the commonest type of atresia. The surgical technic employed at the Children's Memorial Hospital in 36 of the 43 cases of this type has been reported in detail elsewhere.[1, 14] Sixteen of the 36 cases operated are living; 15 of the first 21 patients died, a mortality of 71%, while there have been only 5 deaths in the last 15 cases, a mortality of 33% in this group. Thus, the over-all mortality in the 36 cases is 44%.

Numerous endoscopic problems have arisen in the management of infants with congenital esophageal atresia. Sufficient information is usually obtained from the esophagram made by giving the infant 0.5 to 1 cc of Lipiodol by mouth to establish the diagnosis and generally diagnostic esophagoscopic inspection is unnecessary. However, bronchoscopic aspiration both pre- and post-operatively has been done in most of the infants operated in this series and in some

bronchoscopic aspiration was done during surgery when secretions became excessive. It is of interest to note that three infants originally referred for bronchoscopic aspiration of excessive tracheobronchial secretions were found to have an esophageal atresia, the secretions being the aspirated pharyngeal and regurgitated gastric secretions.

Of greatest endoscopic importance post-operatively is the development of a stricture at the point of anastomosis of the two segments (Fig. 6, *C* & *D*). From a surgical standpoint this is often difficult or impossible to avoid since often the upper portion of the lower segment is extremely stenotic, its lumen being hardly more than a millimeter in diameter. Rarely do the two segments approximate each other sufficiently to allow for sufficient overlap to permit establishment of a long suture line. Therefore if the development of a stricture is anticipated, due either to a narrow lumen lower segment, a short suture line, or infection at the site of anastomosis, it is imperative that a gastrostomy be done and a string be passed through the esophagus to be anchored in the stomach or intestine eventually being brought out through the gastrostomy. Such a string serves as a guide through the irregular, fibrous anastomosis. Gastrostomy, which in early cases in the series was done routinely, greatly facilitates dilatations of the stricture since it is often impossible to find the lumen from above.

At present there are eleven infants or children who have been treated for varying degrees of strictures following repair of esophageal atresia. Two of these were originally operated elsewhere and were referred for dilatation post-operatively. Some of these cases responded rapidly to one or two esophagoscopic dilatations. In the remainder, response to retrograde dilatations has been fairly rapid, but frequent dilatations have been necessary to maintain the lumen.

Two cases presented an additional problem with important endoscopic features. The first, operated elsewhere and referred for dilatation of the esophageal stricture, was found to have a severe tracheal stenosis at the point from which the lower esophageal segment had been resected from the trachea and the opening into the trachea repaired. Respirations were loud, labored and rasping in character and a brassy cough was almost constant and uncontrollable. Examination of the trachea with the infant bronchoscope demonstrated the area of stenosis to be immediately above the bifurcation, with granulation tissue on the posterior wall of the trachea. This area was gently dilated four times before dilatation of the esophagus could be begun. While the airway has improved and the child is now four years of age, frequent upper respiratory infec-

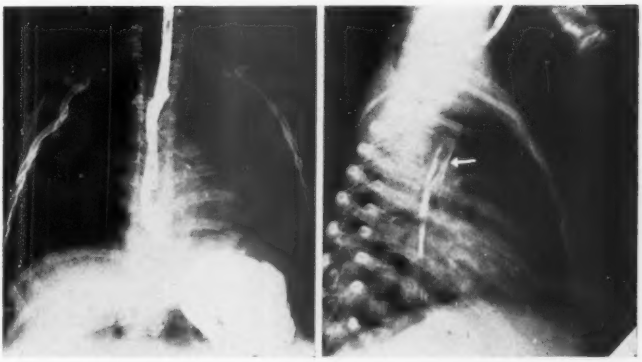

Fig. 7.—Congenital tracheo-esophageal fistula without esophageal atresia. The tract may be seen to descend from the lower trachea to the esophagus.

tions cause an alarming respiratory obstruction and occasional tracheal dilatations are done during the intervals between upper respiratory infections.

2. *Tracheo-esophageal fistula without esophageal atresia:* One infant with this anomaly was seen in 1939. She was 21 months of age and had had repeated episodes of severe cyanosis and bronchopneumonia since birth. It was apparent that while she did not choke or cough during swallowing, cyanosis, violent coughing and pneumonia followed every episode of regurgitation and vomiting. X-rays demonstrated this to be due to a fistula between the lower trachea and the esophagus (Fig. 7). The direction of the fistula, downward from the trachea into the lower esophagus, accounted for the fact that the infant could swallow, but food was aspirated on vomiting. The tract could be visualized in both the trachea and the esophagus and the technic described by Clerf,[15] using sodium hydroxide, was employed to seal the ends of the tract. This was effective for four years when it recurred. The child was operated elsewhere and the fistula closed surgically. This is now without question the procedure of choice although at the time the infant was first seen the external surgical approach was considered too hazardous.

III. ESOPHAGEAL ANOMALIES DUE TO CONGENITAL ANOMALIES OF OTHER STRUCTURES

1. *Obstructions due to cardiovascular anomalies:* The problems of tracheal and esophageal obstructions due to cardiovascular

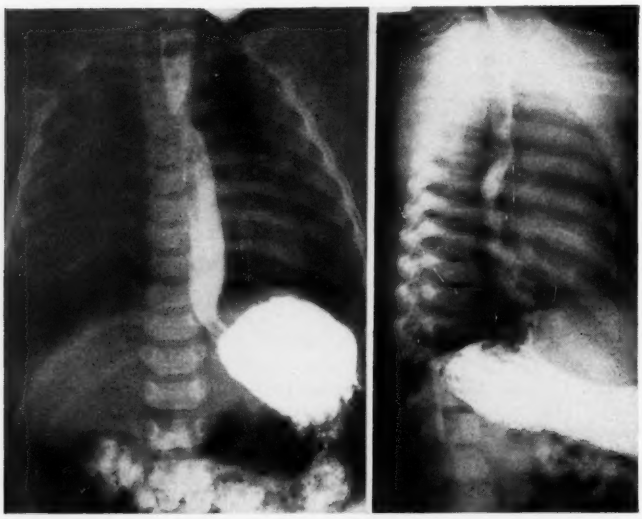

Fig. 8.—Esophageal obstruction due to congenital cardiovascular anomaly, a vascular ring.

anomalies have been reviewed from their endoscopic aspects previously.[16] To this series of cases have now been added an innumerable group to which further additions are made almost every week. They include vascular rings, dysphagia lusoria, and other vascular anomalies of all types which compress the esophagus with varying prominence of the esophageal symptoms. So significant is this aspect that the esophageal picture has become one of the most important features of the diagnostic study (Fig. 8). To this should be added a new diagnostic sign described by Samson[17] and confirmed in two of our cases. It consists of esophagoscopic compression of the esophageal prominence produced by the anomalous vessel and determining which carotid or wrist pulse disappears. Use of this procedure since it was suggested in one infant and one adult in our series has resulted in the disappearance of the right wrist pulse as pressure was applied, indicating that the anomaly is produced by the right subclavian artery originating in the arch of the aorta and crossing the esophagus to go to the right arm.

2. *Lung buds originating in the esophagus:* A lobe of lung arising from the esophagus is a rare congenital anomaly in spite of the common embryonic origin of the esophagus and trachea. One

such case has been observed, and, while it has been reported in detail elsewhere,[18] it is reviewed here because of its endoscopic interest.

S. K., female, five months of age, was admitted to the Children's Memorial Hospital on November 3, 1949. Her weight was 12 pounds and 3 ounces. She was acutely ill with a temperature of 100.6° F., respirations 40 and pulse 120 per minute. She had had respiratory symptoms since birth and x-rays had revealed what appeared to be an atelectasis of the left upper lobe. X-rays at the time of admission were interpreted as showing multiple abscesses of the left upper lobe (Fig. 9*A*). A bronchoscopic examination was made and a large quantity of pus found in the pharynx but after the bronchoscope was passed into the trachea little secretion was present. As the bronchoscope was withdrawn the pharynx, which had been thoroughly aspirated, again filled with muco-pus. Therefore, the esophagus was inspected and at the level of the bifurcation of the trachea a fistula was encountered leading from the left wall of the esophagus through which the pus was pouring. This tract was almost as large as the esophagus itself and was lined by normal mucosa. After appropriate chemotherapy and transfusions bronchograms (Fig. 9*B*) and esophagrams (Fig. 9*C*) were made and the chest was explored. A soft, smooth mass about 8 cm in diameter was found in the apex of the left chest. It was covered with a smooth movable membrane which when opened proved to be the posterior parietal pleura. While dissecting the mass from its bed considerable foul smelling pus was expressed into the esophagus and aspirated. The left main bronchus was firmly adherent to the inferior margin of the mass but did not communicate with it. The blood supply consisted of two fairly large arteries arising from the left pulmonary artery. After these were cut the mass was free except for the large fistula to the esophagus. Thus communication was doubly occluded with nontraumatic ductus clamps and cut. The stump on the esophagus was closed. The postoperative course was uneventful. The pleural cavity drain was removed on the second day, and the retropleural drain was removed on the sixth day. The patient was discharged on the twelfth day after operation. Since then recovery has been rapid and weight gain above average.

Pathology—The gross specimen was covered with a thick fibrous capsule (Fig. 9*D*). Entering one side of the mass was a firm tubular structure 10 mm in circumference, 15 mm long, and containing islands of cartilage. As it entered the mass it bifurcated into two channels which in turn broke up and were lost in the substance of the tissue. The cut surfaces revealed many cyst-like cavities 8 to 10 mm in diameter, containing air, pus and necrotic tissue. Micro-

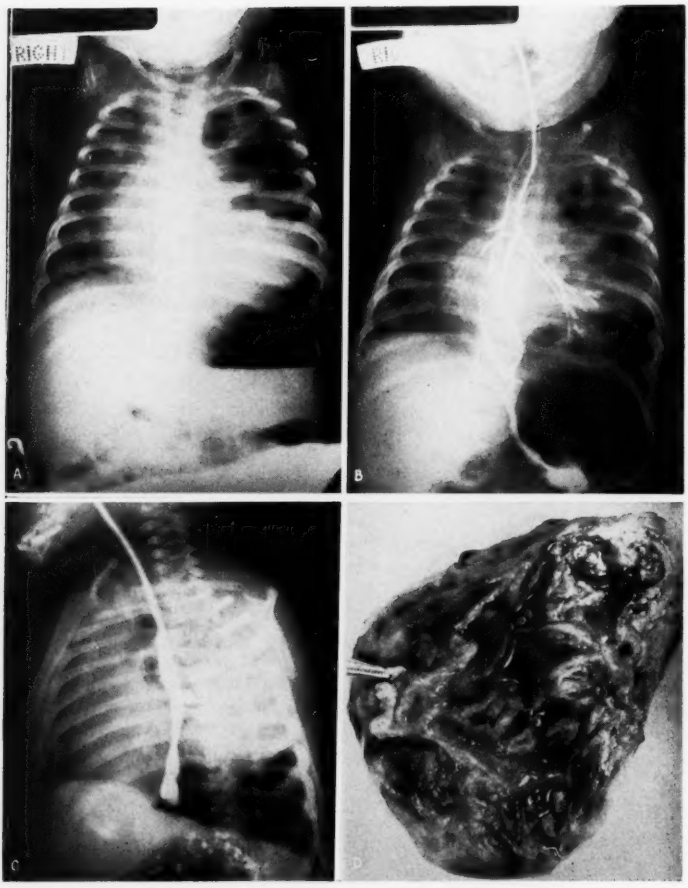

Fig. 9.—Anomalous lobe of lung arising from the esophagus. *A.* Apparent multiple lung abscesses of the left upper lobe. *B.* Bronchogram demonstrating the unusual position and length of the left bronchus. *C.* Esophagram showing the position of the fistula visualized esophagoscopically *D.* The gross specimen of the anomalous lobe with the hemostat attached to the cartilaginous bronchial tract.

scopic examination of the tubular structure confirmed the presence of islands of cartilage and revealed a lining of pseudostratified, columnar epithelium. The tunica propria was composed of collagenous and elastic fibers with marked round-cell infiltration. Smooth muscle fibers were seen, and typical glands such as are normally found in the trachea were present in the submucosa. The mass itself consisted of atelectatic lung parenchyma and bronchioles filled with myriads of polymorphonuclear cells, necrotic material, and occasional vegetable cells. Pathologic diagnosis was ectopic bronchus and lung originating from the esophagus and chronic aspiration pneumonia.

SUMMARY

1. Congenital anomalies of the esophagus are divided into three groups:

   a. Anomalies of the esophagus alone.
   b. Esophageal anomalies with associated tracheal defect.
   c. Esophageal anomalies in association with or produced by anomalies of other structures.

2. The history of dysphagia, aspiration pneumonia, drooling, and "feeding problems" in infants leads to the suspicion of congenital esophageal anomalies.

3. Fluoroscopy with Lipiodol and films suffice to establish the diagnosis in the atresias. Esophagoscopy is needed to assist in the diagnosis of the other anomalies.

4. Therapy of the atresias is surgical. First steps in the therapy of the stenoses is esophagoscopic dilatation. Because of the frequency with which the stenoses can be corrected endoscopically and because of the problems associated with post-surgical strictures, surgery should be contemplated only if adequate endoscopic trial fails.

5. Special endoscopic problems arise in infants with congenital esophageal anomalies. These consist of the pre- and post-operative management of the pulmonary complications and the technical problems of esophagoscopy in the new-born infant. Of particular interest is the management of the strictures of the esophagus and of the trachea that occur in some cases following anastomosis for congenital esophageal atresia with tracheoesophageal fistula.

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DISCUSSION

PAUL C. SAMSON, M.D. (Oakland, Calif.): Mr. Chairman, I am discussing the little sign that Dr. Holinger mentioned in my own paper, and to save time, since we are behind schedule, I would prefer to discuss this sign at that time.

DR. HERMAN J. MOERSCH (Rochester, Minn.): Dr. Jackson, Ladies and Gentlemen: Dr. Holinger has certainly given us one of his usual constructive and brilliant papers, and there is really very little that I can add to what he has said. I should like, however, to re-emphasize two points in his presentation.

First, congenital stricture of the esophagus is a comparatively rare condition, and one that usually is allowed to go untreated before the correct diagnosis is established and the proper treatment is undertaken. Recently, I have seen 2 patients, one a man 34 years old and the other a woman 24 years old, both of whom had congenital stricture of the esophagus. Neither patient had ever eaten solid foods, having always subsisted entirely on liquids, and in neither case had the true nature of the condition been recognized. I both cases dilatation of the esophagus was followed immediately by relief of dysphagia.

Second, with respect to cardiospasm in children, such children often are allowed to go without treatment for an indefinite period before a correct diagnosis is made. It is not unusual to find that their symptoms have been looked upon as functional in nature, and probably as having a basis in some psychologic disturbance. In most cases, cardiospasm in children can be treated satisfactorily by means of the Plummer hydrostatic dilator. However, the results of this treatment on the whole have not been quite so satisfactory in children as in adult persons. This is especially true if the esophagus is markedly dilated, with associated angulation. In cases of this type, surgical treatment may have to be employed.

ALBERT H. ANDREWS, JR., M.D. (Chicago, Ill.): I wish to compliment Doctors Holinger, Johnston and Potts upon their very excellent presentation. Doctor Potts, specializing in pediatric surgery, has made many contributions and especially in the field of chest surgery.

The early recognition of atresia of the esophagus and trachea-esophageal fistula is essential to successful surgery. The nurses in the new-born nurseries must recognize and know the significance of such signs as choking, coughing, gagging and drooling. It is necessary for us to stimulate this phase of teaching in the training of nurses.

DR. JOEL J. PRESSMAN (Beverly Hills, Cal.): I think it must have amazed you, as it did me, to see the enormous size of the stomach that was lying subcutaneously in the chest after the procedure that Dr. Holinger described, and you must have wondered what happened to it when it entered the thorax. It would seem as though it would fill the entire mediastinum and take the place of the heart and both lungs. Actually, an amazing transition takes place when these stomachs are brought into the thorax. They atrophy to such a degree that several I have seen have been smaller than an esophagus dilated from cardiospasm. The stomach therefore adapts itself to its environment at least as far as its size is concerned.

When the anastomosis takes place high up, one of the problems with which we have been confronted is that the junction between the stomach and the esophagus tends less to become stenosed than it does to become angulated. This is probably because of traction upon the stomach, so that a valve-like formation takes place in a manner which doesn't lend itself well to dilatation.

I would appreciate Dr. Holinger's comments on the degree of atrophy he has seen in such stomachs after they are put into the thorax, and also his experience with the obstruction following a high anastomosis from the standpoint of angulation rather than narrowing.

PAUL M. HOLINGER, M.D. (closing): I wish to thank Drs. Andrews, Moersch, and Pressman, for the points that were mentioned. As far as the atrophy of the stomach is concerned, it is interesting to see the manner in which the stomach apparently assumes a smaller size after being placed in the chest. The angulation is certainly of importance and provides one of the problems in swallowing; but the reverse may also occur causing regurgitation upward into the segment of the esophagus which is left. Peptic ulcers develop in this area and produce stenosis later, and add another complication.

Dr. Andrews mentioned the teamwork that is necessary in the management of these infants, and, of course, it is without question a thrilling thing to see the child finally get along after working hours, days and weeks, in the management of such youngsters. Dr. Andrews has been associated with us in many of these cases and realizes full well the problem.

I might mention the fact here that might be of interest, that two of these infants with congenital esophageal atresia had been seen simply because the obstetrician and pediatrician referred the infants for bronchoscopic aspiration, because of the tracheobronchial secretions which were present, not suspecting the diagnos's until having had endoscopic consultation.

Dr. Moersch's remarks with regard to cardiospasm, of course, come from one who knows, and as far as the management of the fourteen year old child, certainly I would be interested to know how he does get along.

LXV

FOLLOW-UP OBSERVATIONS ON THE TREATMENT OF BENIGN STENOSIS OF THE ESOPHAGUS

I. RESULTS OF TREATMENT WITH THE CANNULATED BOUGIE

GABRIEL TUCKER, M.D.

PHILADELPHIA, PA.

II. THE SURGICAL TREATMENT OF ESOPHAGEAL STRICTURES

HERBERT REED HAWTHORNE, M.D. (By Invitation)

PHILADELPHIA, PA.

I. RESULTS OF TREATMENT WITH THE CANNULATED BOUGIE

It was my privilege to present observations on the diagnosis and treatment of a series of cases of non-malignant strictures of the esophagus at the Fourth International Congress of Otolaryngology in 1949. The observations were based on a study of a group of 111 patients observed over a 25 year period at the Graduate Hospital of the Graduate School of the University of Pennsylvania and the University Hospital of the University of Pennsylvania.

Etiologically the cases may be grouped as follows:

	Cases
Congenital stricture (peptic ulcer? web formation)	2
Short esophagus	36
Caustic burns (acid, alkali, iodine, etc.)	63
Benign tumor, with ulceration	1
Ulceration (typhoid, etc.)	7
Traumatic, post-operative	2
Total	111

During this period the treatment used was peroral endoscopic dilatation, with local applications where ulceration was present, and

retrograde and peroral continuous-string dilatation in gastrostomized patients. The results by these methods of treatment were quite satisfactory for the most part. However, there were a number of patients, not over ten percent, who still had difficulty with recurrence of obstruction after a period of apparent cure.

At the San Francisco meeting of the American Broncho-Esophagological Association I presented (with Drs. Lautz, Tracy and Fearon) observations on a series of 14 cases in which evidence of stenosis had recurred and the cannulated bougie had been used for dilatation. The bougie was so devised that it could be used by peroral application, and in gastrostomized cases, could be used either peroral or retrograde with a continuous string.

The period of use of this bougie had not been sufficiently long to give a satisfactory estimate of its value, although results were very encouraging. I requested the privilege of presenting further follow-up observations on this series of cases and I wish to add to it one old case that had suffered a lye burn at six months of age—the patient is now 44 years of age—and at the present time has developed a chronic stricture. This patient previously had had a gastrostomy and had been treated by retrograde bouginage with apparent complete recovery and the gastrostomy had been closed. This stricture which developed in the area of the esophagus previously burned by caustic, proved to be benign and showed a narrowing of the esophagus in the mid-thoracic area at two distinct levels. The patient first developed serious difficulty when drinking orange juice. He swallowed a seed and the esophagus became completely obstructed. Roentgenologic examination with an opaque mixture showed the seed between two stricture levels. This made it impossible to introduce the esophagoscope deeply enough to visualize the foreign body. A stiff expanding-spring forcep was introduced through the upper stricture level by esophagoscopic vision and the seed removed. The indwelling bougie was placed so that both stricture levels were dilated at each application. The stricture, which proved to be benign, responded remarkably well to the use of this indwelling bougie.

The following cases illustrate further the use of the cannulated bougie:

Congenital Stricture. A child 7 months of age, female, had been thought to be normal at birth. Well nourished at the time of admission, she had been regurgitating when attempts were made to give her soft food. Radiographic examination with iodized oil showed a stricture in the upper third of the esophagus and at the lower and middle thirds. Esophagoscopic examination demonstrated the

mucosa of the cervical esophagus was normal with a narrow stricture just at and below the suprasternal notch. A no. 12 F. bougie was passed into the stomach. The lower stricture level could not be visualized. After a few weeks' observation the swallowing had failed to improve, and it was decided that a gastrostomy, the placing of a string through the esophagus, gastrostomy feedings, and bouginage by the continuous-string method should be used. The child was treated by this method, using the old type retrograde bougies which brought the lumen up to a 24Fr. The indwelling cannulated bougies were then used up to a size 34Fr. and at the present time the esophagus is normal and the gastrostomy fistula has been closed.

Acute lye burn. A male child 23 months old swallowed a lye solution; a tube feeding and dilatation of the esophagus in the acute stage of the burn were carried out. Cicatricial stenosis developed at the level of the suprasternal notch; indwelling bougies were then used for retrograde dilatation and carried up to a size 34Fr. The esophagus is normal at the end of a year's observation.

Chronic Stenosis of the Cervical Esophagus. Plummer Vinson Syndrome. A female, 70 years of age, developed a chronic stenosis of the upper cervical esophagus. Indwelling cannulated bougies were used for dilatation, increasing in size up to 34Fr. The patient remained symptom-free for a year but a recent examination showed a slight stenosis had again recurred. A no. 34Fr. cannulated bougie was placed in position without discomfort to the patient. She has maintained the weight gained during the past year and the esophagus is normal.

Post-Irradiation Stenosis of the Esophagus (previously reported). A female aged 40 years came for treatment with complete atresia at the level of the crico-pharyngeus following irradiation treatment for carcinoma of the thyroid. The atresia was relieved by external esophagotomy by Dr. Hawthorne and incision of the stenosed area by transillumination from the pharynx. The cannulated bougie was placed and dilatation carried out up to a size 36Fr. Swallowing was normal. The gastrostomy fistula was closed and the patient is under observation and occasionally swallows a mercury bougie. There seems no tendency to reformation of the stenosis.

Benign Stenosis of the Lower End of the Esophagus. Peptic Ulcer. Male, 45 years of age. Roentgenologic examination revealed a cicatricial narrowing at the junction of the esophagus and cardia. Biopsy was negative for carcinoma. The patient was unable to swal-

low a mercury bougie. Indwelling bougies were used in increasing sizes starting with an 18Fr. size. These were increased gradually to a size 40 and the patient's swallowing returned to normal. This man has gained 25 pounds in weight and roentgenologic examination at the present time shows an adequate lumen through the strictured area. The indwelling bougies were followed by the occasional use of the Hurst mercury bougie up to size 44Fr.

Chronic Cicatricial Stricture. Old lye burn. D. C. aged 15, colored male. This child had swallowed lye at 5 years of age. A severe burn of the esophagus with deep ulceration followed. Gastrostomy was done, a string placed in the esophagus, and retrograde dilatation with a solid bougie was carried out at irregular intervals. The patient was extremely uncooperative, resisting treatment and would not come in for observation. Indwelling bougies were used starting with an 18Fr. The sizes were gradually increased until a 24 size was used and a latex catheter was substituted for the indwelling bougie remaining in the esophagus for one week. A latex catheter size 28 was inserted, the lower end of the catheter being brought out through the gastrostomy opening so that feeding could be carried out both above and below. The string was left in the esophagus alongside the latex catheter. This patient was previously reported as wearing this catheter. He failed to return for a period of a month. When he was finally brought back to the hospital he was swallowing satisfactorally through the catheter but it was found that the upper expanding funnel shaped end of the catheter had become adherent to the cicatricial end of the esophagus. A string had not been placed on the upper end of the catheter and when it was removed it was pulled downward through the esophagus. As a result of this removal the scar in the esophagus ruptured through into the left bronchus, producing a broncho-esophageal fistula. It was decided to treat the patient conservatively—the string being left in the esophagus. The patient was fed entirely by gastrostomy feedings. He was placed on anti-biotics.

Bronchoscopic examination showed a small fistula in the posterior wall of the left main bronchus about ½ cm. beyond the carina which communicated with the esophagus. The boy's general condition remained satisfactory and repeated examination showed the fistula was closing spontaneously. No serious pulmonary complication developed and at the end of a month the fistula was completely closed. Gentle bouginage was then carried out and the cannulated bougie up to size 20 was finally brought retrograde through the esophagus. The patient at this point had become more difficult to handle and finally left the hospital and refused to be treated further by bouginage. He was then turned over to Dr. Hawthorne who

removed the cicatricial area of the esophagus, anastomosing the stomach and the esophagus. Dr. Hawthorne will present further progress of this case.

The use of the indwelling cannulated bougie produces gentle dilatation of the strictured area for a period of hours, instead of minutes, resulting in a stabilizing condition which is much worth while in securing a favorable end result. As the stenosis is dilated the swallowing function returns to normal. The treatment is followed by the use of a suitably-sized Hurst mercury bougie, occasionally, to see that there is not a tendency of the stricture to return. In small children the Hurst bougie in size 30 or 32 is swallowed; and in the adult, the sizes 36 to 44Fr. are selected for swallowing—depending on the size of the individual. If there is difficulty in introducing the bougies at weekly or monthly intervals, esophageal examination is made and if stenosis is found a reapplication of the indwelling cannulated bougie may be used. In cases where there was a tendency to intolerance of the indwelling bougie we have found that the bougie placed was too large and a smaller size was well tolerated, producing a dilating effect by its prolonged sojourn.

In two cases we encountered difficulties that required the skill of the thoracic surgeon. In both patients remarkable results were obtained by Dr. Herbert Reid Hawthorne. Dr. Hawthorne has consented to present the surgical aspects of these cases which resulted in cure.

One very important thing we have learned is that we are dependent upon the thoracic surgeon to help us out when we get into trouble, just as the esophagologist sometimes can be of help to the surgeon.

In certain cases where esophago-gastric or esophagojejunal anastomosis has been performed, a stricture or stenosis may develop. In these instances the indwelling bougie is of value to the thoracic surgeon.

II. THE SURGICAL TREATMENT OF ESOPHAGEAL STRICTURES

The immediate results of an esophageal anastomosis are often excellent but with the lapse of time, some of these same favorable results will require dilatation. The surgeon is fortunate indeed to have the skillful cooperation of the bronchoesophagologist.

I am firmly convinced that it is far better to continue in most instances with observation and esophageal dilatation as indicated throughout the lifetime of the patient with a caustic stricture when the response is at all favorable. Surgery should be employed only

as a last resort. A stricture that responds to dilatation is a far better situation than a post surgical stricture, the scar of which responds poorly to treatment.

Dr. Tucker has described his experience with the 15 year old boy with a caustic stricture of 10 years duration. He mentioned the bronchoesophageal fistula that unquestionably increased the preexisting stenosis. We realized that the resulting periesophageal fibrosis, the result of this infection, would add to the difficulty in the operative treatment. This is in marked contrast to the normal tissues encountered in the tracheo-esophageal fistulas of the newborn. In the case of the boy under discussion we were faced with the problem of a complete stenosis and a gastrostomy life for his remaining years. Through a left transthoracic approach considerable difficulty was encountered in developing a cleavage plane just below, behind and above the arch of the aorta. It required an incision through the beds of the left 8th and 4th ribs and an exposure of the superior mediastinum through an incision along the left sternomastoid muscle to accomplish the separation of the esophagus. The massive amount of periesophageal fibrosis around the upper end of the esophagus was of particular interest, a finding to be considered if surgical treatment of these long strictures is under consideration. The uppermost end of the esophagus was quite firm but with a normal appearing lumen and mucosa. This was determined when the anastomosis was made between the stomach and the proximal inch of the esophagus. The firmness in the wall indicated the pressure of fibrosis, an important matter to consider in the probable increased tendency to fibrosis in an anastomosis with consequent stricture formation.

The post-operative course was uneventful. Figures 1 and 2 show a wide patent anastomosis, three weeks after operation. About five weeks after operation while at home the boy developed meningitis and an interval of six weeks followed before recovery was sufficient to allow a follow-up examination. He was able to take a normal amount of food but Dr. Tucker discovered some narrowing at the line of anastomosis. There is some question as to the narrowing, whether it is mostly due to a valve effect of hypertrophied mucosa or early stricture formation. Dr. Tucker has been able to dilate this without difficulty and is optimistic about the end result. However in retrospect it would have been better judgment to have anastomosed the stomach to the pharynx, where a wider anastomosis could have been formed.

In one of our staged procedures for esophageal atresia and tracheoesophageal fistula a stricture has developed between the anterior skin tube and the gastric stoma. The child is now five years of age

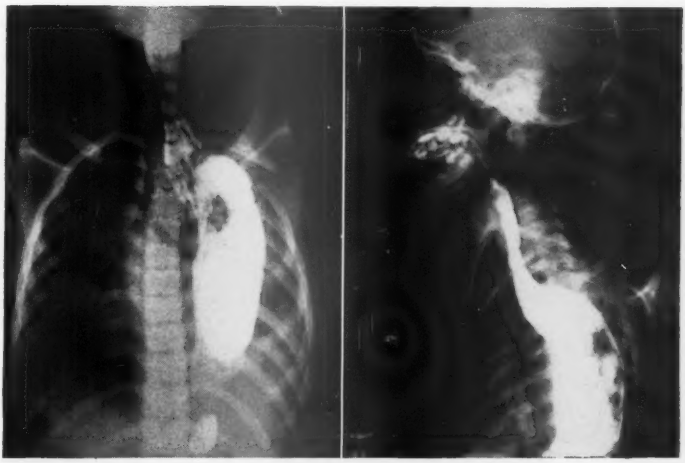

Fig. 1 and Fig. 2.—Antero-posterior and lateral views of anastomosis 3 weeks after operation. Note the wide type of anastomosis that required dilatation at a later date by the Tucker indwelling bougie.

and I believe the uppermost normal esophagus will allow a satisfactory anastomosis; employing the operation I have described—a procedure that is of too great a magnitude in the newborn.

A stricture in the lower esophagus should allow one to go well above the lesion—when surgery is required. A chronic fibrotic process in tissue that is repaired will surely result in a dense scar and contracture. This is of the utmost importance in any type of anastomosis and one must be aware of the fact that a post surgical stricture may be as serious a problem as a stenosis that is being treated by dilatation.

250 South 18th St.

DISCUSSION

Maurice G. Buckles, M.D. (Columbus, Ohio): I wish to congratulate Dr. Tucker on this excellent presentation, also Dr. Hawthorne for his excellent surgical results.

The point I wish to make is that carcinoma arising in the cardia of the stomach may infiltrate the wall of the esophagus in such a diffuse manner that the x-ray pattern may closely simulate that of cardiospasm. We have run into this on three occasions during the past two years. It is simply brought out as a matter of caution in making the diagnosis of benign esophageal obstruction at the distal end of the esophagus.

I feel that if there is any question as to diagnosis, exploratory thoracotomy should be performed by the transthoracic approach as it is easy to handle any condition that may be found, whether it be carcinoma or a benign organic stricture. Esophagoscopy alone in some of these cases does not give the answer, inasmuch as the mucous membrane may be intact.

LXVI

UNUSUAL CAUSES OF DYSPHAGIA

PAUL C. SAMSON, M.D.

AND

DAVID J. DUGAN, M.D. (By Invitation)

OAKLAND, CALIF.

Dysphagia is the commonest symptom of esophageal disease. Statistically, the most frequent causes are mucosal and intraluminal affections. Mural lesions and extraesophageal disease must, however, be considered in the differential diagnosis when swallowing difficulties are encountered. In the cases to be presented, three unusual causes of dysphagia will be discussed.

REPORT OF CASES

CASE NO. 1.—This was a 27 year old Spanish male who was hospitalized in March, 1949. For the previous six months he had suffered from frequent attacks of substernal discomfort and a sensation of choking with deglutition. During the week prior to his admission the patient experienced daily attacks of tightness in the chest and a sensation that food was lodging beneath the sternum. Substernal distress persisted from three to four hours following the ingestion of small amounts of food. The patient did not regurgitate but was nauseated and could not eat until the tightness subsided.

Prior to his present illness, he had been successfully treated for duodenal ulcer and for infectious hepatitis.

Details of the roentgen examination are shown in Figure 1.

At esophagoscopy, a firm mass was seen to be projecting from the anterior wall of the esophagus 30 cm from the incisor teeth. The mass was 6 cm in length and the overlying mucosa was intact. No attempt was made to secure a biopsy specimen. There was neither fixation nor infiltration and the esophagoscope could be passed beyond the mass with ease.

Right posterior thoracotomy was performed and a segment of the fifth rib was removed (D. J. D.) The azygos vein coursed over the tumor and was divided. A Levine tube inserted preoperatively could be palpated through the esophageal wall. The longitudinal muscle was incised over the tumor and the lobulated

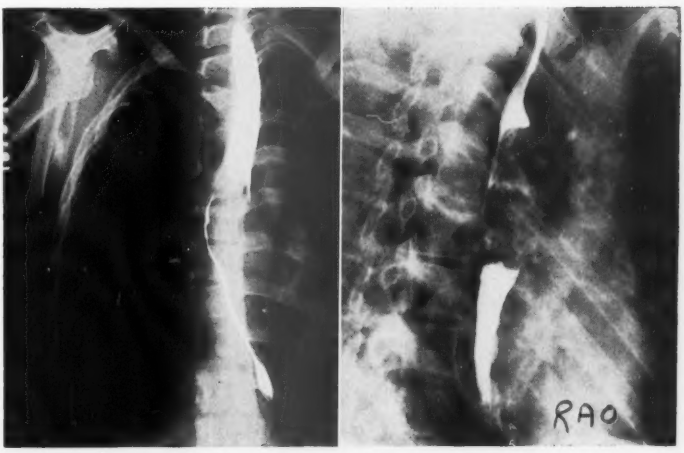

Fig. 1.—With a barium swallow the upper esophagus canalizes normally but dilates. The filling defect is 8 cm in length. The characteristic shelving both above and below is better shown in the oblique projection. In this area the barium column is narrowed but the mucosal relief pattern is intact. The esophagus is normal below the defect. No soft tissue mass is seen.

mass was then easily dissected free leaving the mucosa intact. The longitudinal muscle was re-approximated in two layers with fine interrupted cotton sutures.

The tumor was covered by a thin capsule (Figure 2). The microscopic picture was typical of benign leiomyoma.

COMMENT

The available literature on leiomyomas of the esophagus recently has been reviewed by one of us (D. J. D.).[1] Including the present case, a total of 15 successful removals has been reported. These tumors are uncommon but the roentgen findings are fairly characteristic. The picture is not easily confused with mucosal lesions such as carcinoma or ulceration. Of importance from the endoscopic viewpoint is the decision *not* to procure a biopsy specimen if the overlying mucosa is intact[1, 2, 3] Otherwise, the mucosa may become so adherent to the tumor that with its removal the lumen of the esophagus will be entered. From the surgical standpoint, it is valuable to remember that if these lesions are in the middle or upper portion of the esophagus a right thoracotomy should be performed. If they are found in the lower esophagus, it probably

Fig. 2.—*A.* Benign leiomyoma, roughly fetus-shaped, measuring 6.5 x 2.5 cm. *B.* The tumor has been sectioned, showing a characteristic glistening surface and areas of degeneration with hemorrhage. (Reprinted from Dugan, D. J., and Meagher, T. (1)).

makes little difference whether the chest is opened from the left or from the right. Should there be suspicion that esophageal resection might be necessary, a left-sided approach is superior since the stomach can then be mobilized through the left diaphragm. The use of an indwelling Levine tube permits the operator to be absolutely certain that the longitudinal muscle is divided over the tumor on the opposite side from the esophageal lumen.

CASE NO. 2.—This patient was a female aerial trapeze artist, aged 29. Three months before we saw her there was noted the gradual onset of a gnawing pain beneath the lower sternum which was made worse on eating. In the preceding few weeks the pain had increased and was now almost constant whether eating or not. Recently she had been subsisting on little more than bread and milk. There had been a weight loss of five pounds. On swallowing, the patient had a sensation of food sticking behind the mid-sternum. There was no history of regurgitation or vomiting. A diagnosis of periesophageal hiatus hernia had been made elsewhere.

Three years before, the patient had seen several physicians because of vague pains which radiated across her back and beneath her left breast and were without apparent cause. The pains were sharp in character and were increased both by deep breathing and by exercise. She had always been told that x-rays of her chest were normal.

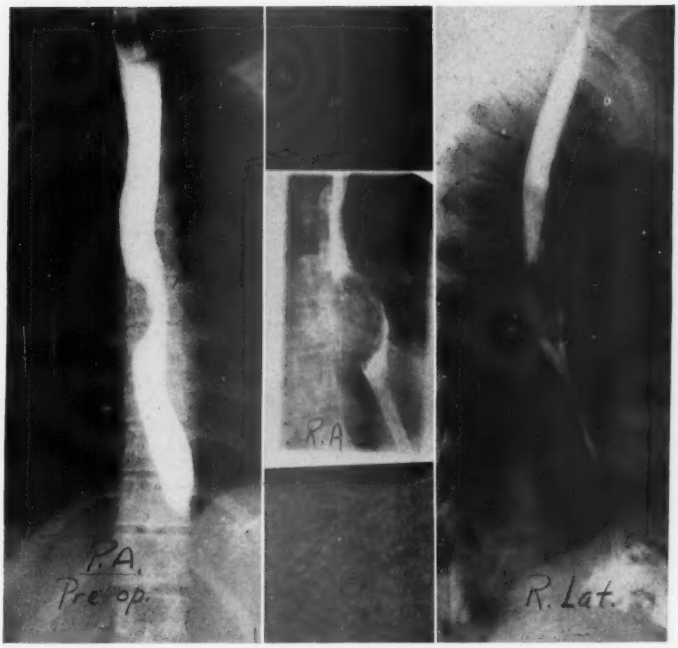

Fig. 3.—The upper esophagus canalizes normally. There is slight delay but no dilatation. Just below the level of the carina there is a scalloping defect impinging on the right anterolateral border of the esophagus without true shelving. In the anterior projection the appearance is that of extra-esophageal pressure. Fluoroscopy and spot films suggest a small ulcer crater posteriorly. The lower esophagus is normal.

Three roentgenograms of the esophagus with barium swallow are illustrated in Figure 3.

On May 15, 1950, esophagoscopy was performed under pentothal-curare anesthesia using the 9-53 instrument. The esophagoscope was passed with ease and on preliminary examination there was little to be seen. With further search, slight mucosal erosion was noted approximately 28 cm from the incisor teeth. Here, there was some bulging inward from the right lateral wall. The bulging was soft and did not pulsate. There was no fixation. Because of the indefinite findings the examination was painstaking and took forty minutes.

The patient reacted slowly and was not fully awake for six hours. At that time her temperature was 101 degrees rectally.

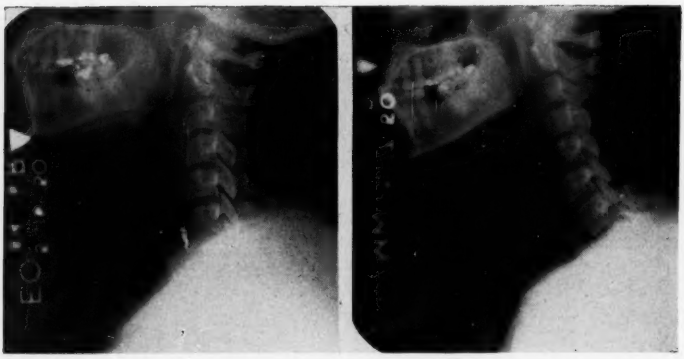

Fig. 4.—*A.* Twenty-four hours following esophagoscopy there is general widening of the prevertebral soft tissue shadow. Swallowed lipiodol has been trapped outside of the lumen in the region of the greatest swelling, pathognomonic of a perforation. *B.* One month later the soft tissue densities are normal.

Twelve hours after the examination the patient complained of discomfort in her upper chest and stated that it was painful to gargle and to swallow. Penicillin, 600,000 units and streptomycin, 1 gram were commenced intramuscularly. Within 24 hours of the esophagoscopy the patient seemed acutely ill and we believed that she had a perforation of the cervical esophagus. She would not flex her neck and could not elevate herself to a sitting position without support to the back of her head. There was exquisite point tenderness along the anterior border of the left sternocleidomastoid muscle without swelling or crepitation. The white blood count was 12,000. X-rays at this time showed widening of the prevertebral shadow and swallowed lipiodol confirmed a small perforation at the level of the sixth cervical vertebra (Figure 4).

Antibiotic therapy was intensified and with sedation and parenteral fluids the patient's condition seemed to improve. During the night, however, the pain and tenderness increased, necessitating demerol for control; by 3:00 A. M. (36 hours after esophagoscopy) the temperature was 102.8° rectally.

Forty hours following esophagoscopy an incision was made along the anterior border of the left sternocleidomastoid muscle. An early retroesophageal abscess was drained and the posterior perforation in the esophagus was closed with fine silk. The fever peaked at 103.8 degrees then rapidly returned to normal in 48 hours. The

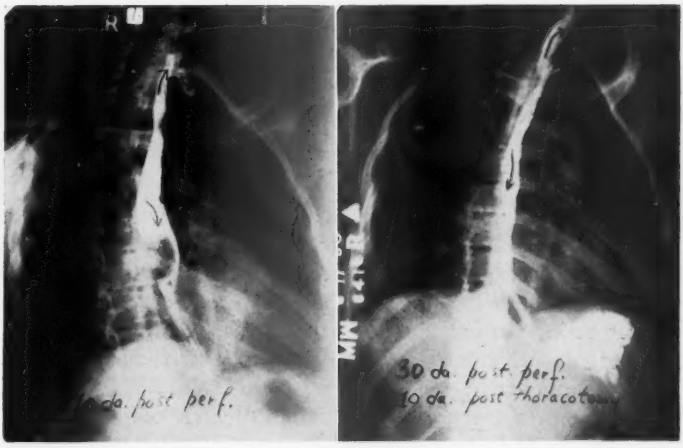

Fig. 5.—*A.* Ten days following cervical mediastinotomy and repair. The upper arrow outlines the short postperforation sinus tract which is asymptomatic. The lower arrow shows the filling defect previously described. *B.* Ten days following thoracotomy. The sinus tract has become obliterated and the esophagus now fills normally.

patient was started on fluids by mouth on the third day and was discharged on the sixth postoperative day.

Twelve days later the patient was able to swallow soft food satisfactorily. The roentgenogram showed a persisting small sinus track at the site of the perforation but this disappeared within three weeks' time (Figure 5).

The patient's original complaints of dysphagia and odynphagia did not abate. Right posterior intercostal thoracotomy was performed three weeks following esophagoscopy (P. C. S.). At the level of the fifth rib posteriorly a mass was found in the region of the esophagus which was partially nodular and partially fluctuant. The esophagus was dissected free above and below the region and it was obvious that there were firm inflammatory adhesions to the posterior hilum. On further dissection an abscess cavity was entered which contained one ounce of thick, odorless pus. Gram stains showed no pyogenic organisms. The abscess was anterolateral to the esophagus and indented the wall. It was evacuated and obviously was connected with conglomerate soft enlarged lymph nodes which were in the subcarinal region. With probing, caseous material extruded. The irregular mass was totally removed and measured 4 x 2.5 x 2 cm. The fibrous wall of the abscess was likewise

completely dissected from the esophagus. The mediastinum was drained extrapleurally by means of a Penrose drain through a separate paravertebral stab wound and the mediastinal pleura was closed. The pleural cavity was drained by an intercostal water-seal tube. The convalescence was uneventful and the patient was discharged from the hospital with an expanded lung and a solid wound ten days following operation.

Pathologically, the wall of the abscess exhibited no characteristic pattern. The lymph nodes showed caseation necrosis surrounded by epithelioid cells; a few giant cells of the Langhans type were encountered. Both the pus and the lymph nodes were subjected to acid-fast stain, culture and animal inoculation. A few acid-fast bacilli were identified on direct smear but neither fungi nor pyogenic organisms were recovered. Cultures and guinea pig inoculations failed to confirm the presence of viable tubercle bacilli.

As a prophylactic measure immediately after surgery, the patient was placed on streptomycin and PAS therapy. She was kept strictly in bed for three months and started back to work in six months. Over a two months' period, 24 grams of streptomycin were given and over a three months' period approximately 1100 grams of PAS.

She has remained well and is now back at work as an aerialist. The thorax is clear, there have been no swallowing difficulties and no return of the vague pleurisy-like pains from which she originally suffered.

COMMENT

Separate remarks concerning the essential pathology, and the endoscopic complications are in order.

Cases of this type do not seem to be common. In 1940, before this Association, Clerf[4] presented a patient whose periesophageal tuberculous abscess evacuated itself into the lumen and a cure resulted. Jacob[5] reported improvement in dysphagia following radiation therapy directed at presumably tuberculous mediastinal glands. Apparently in only one other reported case has surgical excision of lymph nodes been carried out.[6]

Undoubtedly, the patient's previous pleurisy-like pains were due to the chronic lymphadenitis since this complaint has not returned. Enlarged subcarinal tuberculosis lymph nodes are not an uncommon finding during resection in tuberculosis and so it is possible that the dysphagia was due to the more recent development

of the abscess with accompanying inflammatory deformity and fixation of the esophagus.

The diagnosis of tuberculosis must be presumed because of the findings of acid-fast bacilli and the characteristic microscopic picture. Bacillary viability is not so certain, however, because of the inability to recover the organisms through culture and guinea pig inoculation. These observations lend validity to the findings of Griffith[7] and of Feldman and Bagenstoss[8] who showed that viable tubercle bacilli rarely were found in retrogressive infections, old encapsulated lesions or in sclerotic lymph nodes. Nevertheless, it was felt wise during the post-operative course to insist on a period of bed rest and to administer antimicrobial drugs. It is possible that mere drainage of the abscess might have sufficed. It was obvious, however, that the lymph node mass and the abscess were one pathologic process; surgical justification for adenectomy lay in the fact that the inferior nodes were necrotic and formed a portion of the wall of the abscess.

Endoscopic perforations of the esophagus are unhappy events. One of us (D. J. D.)[9] recently has reviewed our personal and consultative experience with these lesions. In the past four years we have seen eleven cases; six of these were cervical perforations. Contrary to some reports it is our considered opinion that cervical perforation usually is not the result of damage from the advancing tip of the esophagoscope but most frequently occurs during a relatively long procedure as a result of pinching the posterior wall of the esophagus between the rigid tube and the anterior curve of the cervical spine. In one instance, for example, a cervical perforation occurred exactly over hypertrophic anterior spurs on the sixth cervical vertebra.

In an acutely ill patient, there is a triad of symptoms which we believe to be pathognomonic of endoscopic perforation of the cervical esophagus: (1) severe localized pain on swallowing or gargling; (2) severe irritation of the sternocleidomastoid muscle which results in (a) resistance to flexion of the neck and (b) characteristic support of the back of the head with the hand in changing from supine to sitting position; (3) exquisite point tenderness along the anterior border of the sternocleidomastoid muscle with or without the presence of swelling or crepitation.

The proper treatment of these lesions has been the subject of some controversy. There has been considerable support for conservative treatment with intensive antibiotic therapy, reserving surgery for failures on this regimen. Certainly this has been successful

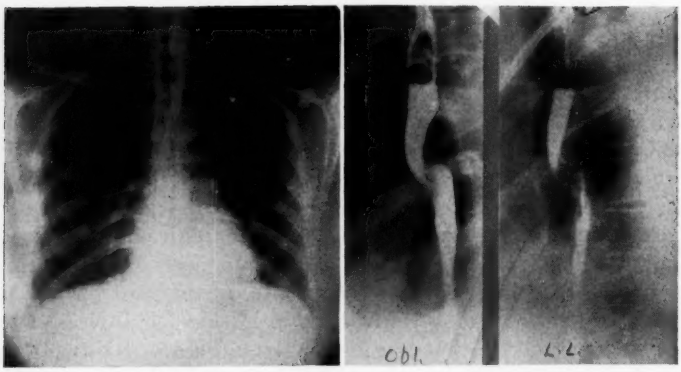

Fig. 6.—In this anterior view there is apparent narrowing and slight displacement of the barium-filled esophagus just above the aortic arch. There is little or no dilatation of the upper esophagus.

Fig. 7.—Spot films in both oblique and lateral projections show compression of the esophagus from behind. The extrinsic pressure is applied in a characteristic spiral which ascends from left to right over a length of 3 cm. The mucosal relief pattern is normal.

in a number of cases. Likewise it has failed and, unfortunately, the surgeon may then be faced with a mediastinal phlegmon with which it is impossible to cope. Based on our own experiences we now strongly advise operation as soon as the diagnosis is made. Hours are important and if the operation can be performed within 12 to 24 hours of the accident the chances for success are greatly enhanced. If the perforation is large enough to be visualized by a swallow of lipiodol it can be located at operation and closed with fine silk. During the stage of contamination (circa 12-18 hours) these sutures will hold but it actual invasive infection of the esophageal wall has taken place, leakage is common.[10] In retrospect, we vacillated unduly in the decision to operate on this patient.

Case No. 3.—This was a white, married female, aged 29. For approximately 18 months the patient had complained of difficulty in swallowing and a sensation that food would stick behind the upper sternum, before passing onward. There was no regurgitation or vomiting. The patient had a constant desire to swallow and frequently gagged when not eating. Occasionally she suffered severe coughing spells while attempting to clear her throat. The patient denied paresthesia of the right arm nor did she notice any differences in temperature of the two arms. The physical examination revealed

no pathology. The blood pressures in the two arms were essentially the same, 106/70.

Roentgen examination with barium swallow revealed a characteristic defect (Figs. 6 and 7).

Under pentothal anesthesia, esophagoscopy was performed and 25 cm from the incisor teeth a pulsating area was noted posteriorly. The overlying mucosa showed minimal congestion. Backward pressure on this area with the beveled opening of the tube completely eliminated the right radial pulse but did not affect the left radial nor either of the temporal or carotid pulses.

At left posterolateral thoracotomy the fourth rib was removed subperiosteally (P. S. C.). The aortic arch, the left common carotid, and left subclavian arteries were normal in position and distribution. Two centimeters beyond the origin of the left subclavian vessel was a large artery which arose from the descending portion of the arch at an acute angle and plunged at once into the posterior mediastinum. It was found to course upwards and to the right behind the esophagus. Pressure on this artery obliterated the right radial pulse. The vessel was ligated and suture-ligatured both proximally and distally, then divided. The distal stump retracted well to the right of the esophagus.

COMMENT

Anomalies of the aortic arch were first attacked surgically when Gross[11] in 1946 reported the successful ligation of an anomalous right subclavian artery for dysphagia lusoria in an infant.

It is of interest that this anomaly was first described more than two centuries ago, apparently by Hunald[12] in 1735. The term "dysphagia lusoria" is attributed to Bayford[12] who, in 1794, described the condition in an adult. To our knowledge, however, only one instance of surgical correction of this abnormality in an adult has been reported.[13]

Including the case above, we have now made a clinical diagnosis of anomalous right subclavian artery in three adults although we have operated on but one. In two, esophagoscopy has been performed and the characteristic pressure sign as described above was present. If positive, this sign should be pathognomonic of the condition without resort to diodrast studies.

In 1948 Holinger, Johnson and Zoss[14] gave an excellent presentation before this Association of the bronchoscopic findings in eight patients who had air way obstruction from congenital cardio-

vascular anomalies. While they were not described, we wonder whether similar signs of pulse obliteration might not be elicited from gentle pressure on pulsating tracheal or bronchial lesions. If this is possible, careful check of the radial, carotid and temporal pulses should give a better lead as to the type of anomaly present.

SUMMARY

Some unusual causes of dysphagia have been illustrated by means of three case reports. In each instance the endoscopic findings have been correlated with the diagnosis made at thoracotomy.

In the first patient, a benign leiomyoma of the esophagus was removed.

In the second patient, dysphagia was due to chronic tuberculous lymphadenitis with localized abscess formation. Surgical excision of lymph nodes and abscess was performed. In this patient, likewise, discussion has been given regarding the diagnosis and treatment of endoscopic perforations of the cervical esophagus.

The third patient suffered from dysphagia lusoria. At operation the anomalous right subclavian artery was easily ligated and divided.

A new esophagoscopic sign has been described.

We have briefly discussed the possible application of this principle, the endoscopic pressure-obliteration of various peripheral pulses, in the bronchoscopic and esophagoscopic diagnosis of other anomalies of the aortic arch.

2938 McCLURE STREET.

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DISCUSSION

DR. WHALEN (Stockholm, Sweden): I have done some investigations of mucosal infections in patients who had dysphagia and have divided them into three stages:

In the first stage it is impossible to make the differential diagnosis. In the second stage you have an alteration of the lumen and irregular contour. In this stage it is still impossible to make a differential diagnosis. In the third stage, months, and even years, later, constriction and dilatation is more pronounced, so that it is impossible to treat the patient with intra-oral catheterization. The prognosis is therefore very serious, and in a vast number of our cases, it has been necessary to do gastrotomy.

C. L. JACKSON, M.D. (Philadelphia): I would like to say apropos of this type of case, I saw my first one in the last year or two. In this case I had the good sense also not to bite through the normal mucous membrane. I think that point is worth emphasizing. If you have a rounded mass that simply bulges into the esophagus, but with an unbroken surface, it is better to open the chest and get in from the outside without doing a biopsy. In our case, as in Dr. Samson's, the surgeon, Dr. George Rosemond, was able to shell out the tumor without having any trouble with adhesions to the mucous membrane, as had been reported in some previous cases where a biopsy was done. So do not be too ambitious to bite into everything you see through the esophagoscope.

DR. PAUL H. HOLINGER (Chicago, Ill.): I simply want to add to Dr. Samson's very excellent presentation that we used his sign in two of our patients, one an infant who had an obvious tracheal obstruction, and on passage of the scope through the area of stenosis in the esophagus, residents were palpating the pulses on the wrists and in the neck, and the pulse in the right wrist disappeared. The vascular anomaly of the right subclavian artery was confirmed on surgical exploration.

The second patient is an adult who has been a problem as far as diagnosis is concerned. She has both a tracheal and esophageal compression, which we photographed. The same procedure was used, and startlingly enough, the pulse in

the right arm completely disappeared as the scope went through this area. It is a very valuable sign, I feel, and one which appears to be safe enough to use.

DR. PAUL C. SAMSON (closing): I wish to thank Dr. Whalen, Dr. Holinger, and Dr. Jackson, for their kind remarks.

The speed and efficiency with which one functions in Chicago is well illustrated by this discussion of Dr. Holinger's. We talked about this little sign of mine, I think, exactly sixteen days ago here in Atlantic City at Haddon Hall at the meeting of the American Association for Thoracic Surgery. He said, "I have just the case to try it on," and he goes home, and I understand he has already made a movie of it.

LXVII

PORCUPINE QUILLS AS LARYNGEAL FOREIGN BODIES

REPORT OF TWO CASES

FREDERICK T. HILL, M.D.

WATERVILLE, ME.

While these case reports rightly do not come within the province of Broncho-esophagology, they do add to the already extensive list of foreign bodies found in the food or air passages. No technical problem was involved in their removal and interest lies solely in the type of foreign body and the manner of invasion. Both patients were adults and, as might be expected, were wearers of full upper dentures, which so frequently renders the individual insensible to an undesirable element in his food or drink.

CASE 1.—A woman, aged 58, a farmer's wife from some forty miles distant, was seen because of marked dysphagia of three days duration. While this came on after eating she had no recollection of having swallowed anything which might account for it. The morning of the examination she had raised some blood. Inspection with the laryngeal mirror revealed a slender, spinous, foreign body projecting from the tip of the epiglottis, it having been driven downward underneath the mucous membrane to its base. Except for a moderate surrounding area of inflammatory reaction the larynx appeared negative. While removal required considerable traction this was easily accomplished by the indirect method under local anesthesia. The foreign body, 2.5 cm in length, was promptly identifed by the patient as a porcupine quill. Her only explanation of how she happened to swallow it, was that the dog had brought a porcupine into the kitchen and "it must have got mixed up with the food."

CASE 2.—A telephone linesman, aged 23, was referred by his local physician because of marked pain on talking or swallowing of one day's duration. This followed drinking from a water-jug while working on a cross-country line through a wooded section. Mirror examination revealed a long slender foreign body lying obliquely horizontal in the supra-glottic region with its tip implanted in the posterior wall of the hypopharynx. With each act of swallowing it

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From the Thayer Hospital.

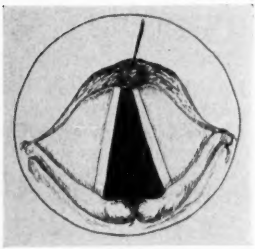

Fig. 1.—View of Larynx showing porcupine quill projecting from tip of epiglottis.

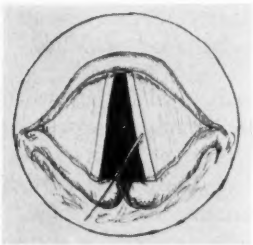

Fig. 2.—View of Larynx showing porcupine quill imbedded in posterior wall of hypopharynx and extending across glottis.

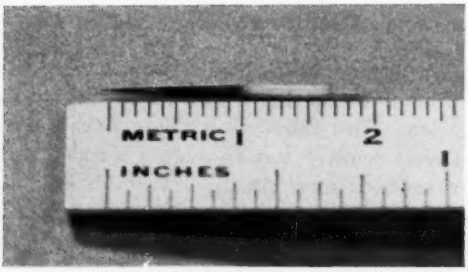

Fig. 3.

seemed to stab a little more deeply. Again using the indirect technique, the foreign body was removed with side-grasping forceps of the McKenzie type. By now, being porcupine-conscious, I had no difficulty in identifying the intruder. How the quill got into the water-jug could not be explained.

The North American porcupine is a rodent of the genus Erethizon. It has sharp, erectile, readily detachable spines or quills, mingled with its hair. These quills are sharp as needles and are tipped with minute barbs which point backward and when impaled in human or animal flesh, tend to rip it on withdrawal. These barbs are such that even slight muscular movements cause still deeper penetration; so that if not removed, the quill may become an invader of dangerous potentialities.

While the ingestion of porcupine quills is extremely rare it seems fortunate that they tend to implant themselves firmly in any adjacent tissue and thus remain in an easily accessible region. Otherwise initial lodgement in the esophagus or the bronchus might present complications, due to their penetrating characteristics, unless discovered early and removed. In the second case the quill appeared almost teetering on the brink of the glottis, yet removal required considerable traction.

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LXVIII

SUPERIOR VENA CAVAL OBSTRUCTIVE SYNDROME

REPORT OF THREE CASES

WILLIAM A. LELL, M.D.

PHILADELPHIA, PENNA.

Although there is considerable question as to the first authentic report of this clinical entity, Hunter's description of a case of superior vena caval obstruction from an aortic aneurysm, observed at autopsy in 1757, would seem the most authentic. Since that time sporadic cases have been reported and Fischer,[1] in 1904, collected 252 cases from the literature. Various authors reported 40 additional cases between 1904 and 1934.

Between 1934 and January, 1946, McIntire and Sykes, Jr.[2] collected an additional 111 cases, 56 of which were proven by autopsy. The work of these authors not only gives an exhaustive review of the literature but also a most excellent discussion of this subject. In the January, 1951, issue of Diseases of the Chest, McCord, Edlin and Block[3] have reported a series of 11 cases of superior vena caval obstruction during a period of one year, and call attention to the possible clinical studies that may aid in the diagnosis. They stress the importance of chest phlebograms for reliable information concerning the location of obstruction. The cases these authors reported showed interesting etiological factors. More recently Ferguson and Werner[4] also reported 2 cases in which the vena cava was involved by adjacent tumors.

In spite of the fact that obstruction of the superior vena cava is supposed to be a rare clinical entity, I had the unusual opportunity of seeing 3 cases with such involvement within a period of two months. All three patients presented the typical finding of a gradually developing edema of the neck and fact with subsequent extension to the arms and hands, the right side of the body showing the more marked fullness. The patient experiences a tight compressing feeling of the upper chest associated with a varying degree of dyspnea and cyanosis of the lips, ears and face. Another constant finding is the distended prominence of the veins of the neck, with many grape-like clusters of telangiectatic venules dispersed over the front of the chest down to the xyphoid process. The rapidity with

which these changes progress depends entirely upon the degree of encroachment upon the superior vena cava and the location of the lesion in relation to the vena azygos. When both these vessels are involved the edema occurs with lightning rapidity, and these unfortunate individuals present a sad spectacle to behold.

Thrombosis of the superior vena cava by indirect injury has been considered a common etiological factor; however, in all three of the cases here reported it was directly due to infiltration of the vessel by adjacent malignant masses.

Of particular interest in these cases was the fact that although they all presented typically the same objective visible changes, each one offered a puzzling diagnostic problem as to the etiological factor, until exploratory thoracotomy revealed the true cause.

REPORT OF CASES

CASE 1.—A. G., a 45 year old Italian male was first admitted to the Abington Hospital on September 20, 1950, because x-ray studies made by Dr. Robert P. Barden, at Chestnut Hill Hospital on September 16, revealed a large, irregular tumor mass extending into the right chest from the superior mediastinum and hilar region, with irregular ventilation of the entire right lung, indicating pressure upon the right main bronchus. This was particularly marked in the right upper lobe, where there was obstructive emphysema involving the anterior segment.

A bronchoscopic examination was made on September 21, 1950, which revealed the motility of the larynx to be normal. The trachea was of normal caliber and there was no ulceration along the right tracheal wall, nor was there any apparent fixation of these structures. The bifurcation was not thickened. The lumen of the right main bronchus was not altered in size. The right upper lobe orifice was clearly visualized and this did not show any fungating tissue to be projecting from it. The remaining portion of the right main bronchus showed no evidence of any ulcerating tissue. By using a curved aspirator secretion was aspirated directly from the right upper lobe opening, and this was saved in sterile containers and one specimen was submitted for cytologic study and the other for Tb and bacteriological study. Examination of the left main bronchus did not reveal any gross abnormality. In spite of the fact that we were unable to find any extension of this lesion directly into the tracheobronchial tree it was felt that the most likely pathology here was a malignant process, possibly arising from the mediastinum, in the hilar area of the right lung.

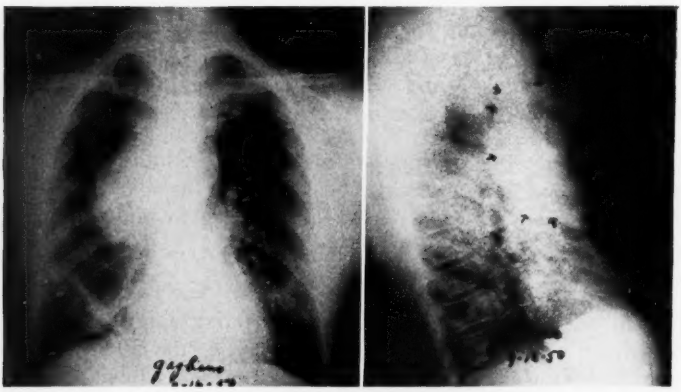

Fig. 1.—Roentgenograms, Case 1, A. G.

The studies of the bronchial secretions showed no acid fast bacilli in the Tb smear, and the bacteriological culture showed no growth after forty-eight hours. The cytologic study was negative for tumor cells.

Because of the nature of the lesion exploratory thoracotomy was felt indicated and the patient was admitted to Pennsylvania Hospital on the service of Dr. John B. Flick, on October 8, 1950. His chief complaint was dyspnea, aggravated by sudden movement or change in position. He had noted that his neck had progressively become more swollen, and he complained of an irritating dry cough of five to six weeks' duration. For the past two weeks he had noticed a huskiness to his voice, which he never had had before. There was no dysphagia present.

Physical examination revealed bull-like fullness of the neck with some edema of the entire face. The right eyelid was definitely puffy, the left not so. Prominent venous arborization was clearly visible over the entire chest. The veins of the arms also appeared engorged and remained so when the arms were elevated.

Laboratory studies showed a normal blood count; negative urinalysis; urea nitrogen 11; total protein, 5.9; albumin 4.4; blood sugar 94 and negative Wassermann.

Repeat x-ray examination again revealed a large tumor mass arising from the right hilum and extending anteriorly and superiorly, and judging from the irregularity of the tracheal shadow this appeared to compress the trachea itself. The right diaphragm was

slightly elevated and moved with much restriction, and there was marked paradoxical motion with the sniff test, confirming phrenic paralysis. There was marked obstructive emphysema in the upper portion of the right upper lobe, with formation of large emphysematous bulla. The radiologist was also of the opinion that this was a malignant lesion (Fig. 1).

On October 12, 1950, Dr. John B. Flick performed an exploratory thoracotomy on this patient. The pleural cavity was opened in the bed of the fifth rib. The pleural cavity contained about 100 cc of clear, amber fluid. The upper lobe of the lung was adherent to the chest wall by string like adhesions. These were divided and some were ligated with 0000 black silk. The upper lobe of the lung, particularly the apex, was the site of numerous subpleural blebs, some of which were 6 to 7 cms in diameter. The fissure between the upper and middle lobes was poorly developed. The fissure between the upper lobe, middle lobe and the lower lobe was well developed. There was a hard mass, approximately 9 cms in diameter, which seemed to surround the hilum of the upper lobe and extend into the mediastinum. This mass was adherent to the pericardium and completely obscured the superior vena cava. The azygos major was distended. The right half of the diaphragm was high and did not move with respiration. No lymph nodes were palpable in the pulmonary ligaments. It was obvious that the lung was not resectable, and it was not certain whether the mass originated in the lung and spread to the mediastinum or originated in the mediastinum and spread to the lung, but it was thought the former was most likely.

Biopsy of the tumor mass was made, and the histologic examination was reported as undifferentiated carcinoma.

This patient was given x-ray therapy, with temporary improvement, but the latest follow up revealed that he had developed metastatic extension to the right hip.

CASE 2.—L. D., a 46 year old white male, was referred to the Pennsylvania Hospital on March 22, 1950, by Dr. Howard Hebble, because of hoarseness and intermittent sore throat, of three months' duration. For the past six months he had also had a productive cough with whitish sputum; no dysphagia, but moderate shortness of breath noticed on exertion. He had never had any edema of face or extremities.

Physical examination revealed a very ruddy complexion, but no edema of the neck or face, and no edema of the extremities. Lungs were clear to percussion and auscultation, except for slight

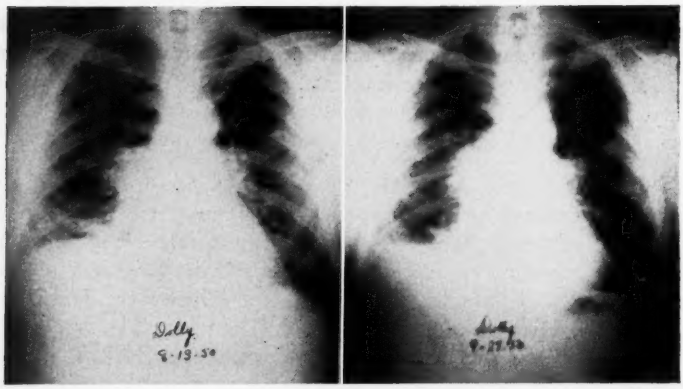

Fig. 2.—Roentgenograms, Case 2, L. D.

diminution of breath sounds in the right base. Heart sounds were normal. Patient had definite hypertension of 188/120.

Mirror examination of the larynx revealed marked polypoid hypertrophy along the posterior half of the right cord. The left cord showed only inflammatory changes.

Laboratory studies revealed a negative urinalysis and blood counts were normal. The Wassermann and Eagle were negative. Blood sugar was 108; urea nitrogen 10; total protein 6-8; albumin 5.0; B.S.P. 12%.

X-ray of chest on March 23, 1950 was reported by Dr. Paul Bishop as suspicious of a bronchogenic new growth with involvement of the phrenic nerve.

Direct laryngoscopic examination on March 24, 1950 revealed both cords to be quite thickened, particularly the right, on the posterior aspect, and the excess tissue along the margin of the right cord was removed and studied histologically, and found to be keratosis.

Bronchoscopic examination revealed no changes endobronchially suggestive of new growth. The cytologic examination of the material aspirated was negative for tumor cells.

The patient was discharged from the hospital on April 3, 1950 and followed as an outpatient, at which time he showed marked general improvement and his hoarseness totally disappeared.

This patient was readmitted to the Pennsylvania Hospital on June 20, 1950, on the service of Dr. Leonard W. Parkhurst, because of progressive dyspnea and beginning edema of the lower extremities. Studies at this time were essentially the same as on the first admission. With proper rest and medication he showed improvement and was discharged on July 8, 1950.

On September 26, 1950, he was again admitted because two weeks prior to admission he had an upper respiratory infection with cough, productive of yellow phlegm. He noticed that his eyelids started to swell at this time. He also became aware that the veins over his chest were dilated. His ankles and hands became swollen with the slightest activity. Edema of face and extremities became severe on exertion and he had considerable dyspnea.

Physical examination at this time revealed blood pressure to be 130/75, pulse 100, respirations 24. There was marked swelling of the face and neck. Both eyelids were puffed. The skin was definitely cyanotic; the lips were purplish. The right forearm and hand were very swollen; the left hand was slightly so. The skin of the chest from the clavicles to xphoid and costal margins showed clusters of venous capillaries resembling bunches of small grapes. There was no edema of the chest nor any signs of subcutaneous emphysema.

Laboratory studies at this time showed—urinalysis, no albumin or sugar. C.B.C.—Hgb. 14.6 grams, RBC 4.8, WBC 12,500. Total protein 5.7; albumin 4.0; urea nitrogen 10; B.S.P. 10%.

X-ray examination of the chest on September 27, 1950, was reported by Dr. Paul Bishop as follows:

"There is definite increase in the size of the nodular lesion in the right hilum since our examination of July. While not marked this is quite definite and there is also formation of a slight amount of pleural effusion, obliterating the costo-diaphragmatic sulcus. The infiltration in the lower lobe is about the same, partially hidden behind the diaphragm in the diaphragmatic gutter. This diaphragm is still elevated and motionless. The appearance of a superior vena cava syndrome supports the diagnosis of possible neoplasm (Fig. 2).

The swallowing function shows no evidence of any intrinsic lesion; no compression or displacement of the esophagus."

A bronchoscopic examination was made on October 2, 1950, which revealed the lumen of the trachea to be of normal caliber. The mucous membrane was quite congested throughout. The bi-

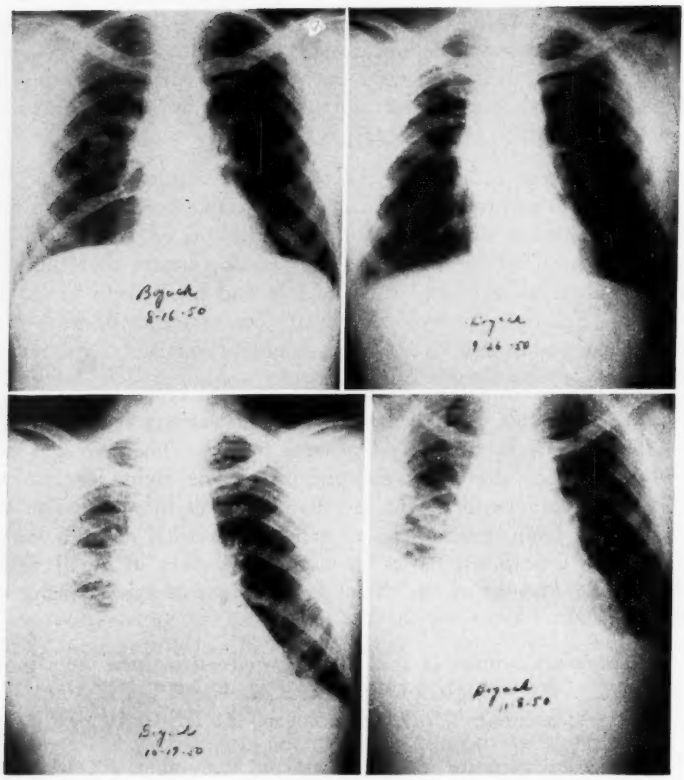

Fig. 3.—Roentgenograms, Case 3, A. B.

furcation was slightly thickened; the mucous membrane here was very fiery red, but there was no evidence of any ulceration. The lumen of the right main bronchus appeared to be of normal caliber throughout. Again the mucosa was moderately hemorrhagic, but there was no evidence of any ulceration. The lower lobe subdivisions showed some changes suggestive of thickening, but no fungating tissue or ulceration could be seen in these structures. Some secretion was aspirated directly from the lower and middle lobe openings and this was saved and sent to the laboratory for tumor cell study. The left side showed the same type of inflammatory changes in the mucous membrane but again no evidence of any ulceration could be seen. As far as we could see by direct inspection there was no evidence of any changes within the trachea or right main bronchus

suggestive of an infiltrative new growth. I could see no evidence of any actual compression of the bronchus itself.

The cytologic study of the material aspirated bronchoscopically was again reported negative for tumor cells.

In view of the patient's progressive clinical course and the more recent x-ray findings, in spite of the fact that the cytologic study of the bronchial secretions was negative for tumor cells, it was felt that this patient most likely had a mediastinal tumor compressing the superior vena cava and exploratory thoracotomy was thought advisable.

Dr. John B. Flick explored this patient on October 21, 1950, and found a hard mass, approximately seven centimeters in diameter, in the medial aspect of the right upper lobe above the bronchus, but not encroaching upon it. This mass extended into the mediastinum, obscured the superior vena cava, and infiltrated the pericardium. The vena azygos major and its tributaries were distended. Since the tumor was not resectable a biopsy was obtained and the chest closed. The histologic report on the tissue was undifferentiated carcinoma.

The patient was placed on irradiation therapy with slow but remarkable progressive improvement, and is still living today.

CASE 3.—A. B., a white male, 42 years of age, was admitted to Pennsylvania Hospital on September 8, 1950. He had been previously x-rayed at the Abington Hospital on August 16, 1950, and Dr. J. Donald Zulick's report of the x-ray examination of the chest was indeterminate.

Studies of the sputum were negative for tubercle bacilli and the cytologic study of the sputum was negative for tumor cells.

On admission to the Pennsylvania Hospital he gave a history that four weeks prior to admission he began having severe pains in his right chest anteriorly, which were knife-like in severity, and would extend posteriorly to the right scapula. This pain subsided in intensity on bed rest of several weeks. During this time the patient had an irritating cough, non-productive in nature. He also experienced general weakness, shortness of breath, and a low grade elevation of temperature during the period of bed rest.

Laboratory studies revealed—negative urinalysis; blood sugar 85; urea nitrogen 10; C.B.C.—RBC 3.9, Hgb. 76%, WBC 12,500, Polys. 72, Lymphs. 21, Eos. 4, Monos. 3.

Bronchoscopic examination was made on September 8, 1950, which revealed the motility of the larynx to be normal; the mucous

membrane quite inflamed. The trachea was of normal caliber throughout; the mucosa here was also quite inflamed. The bifurcation was not thickened. The right upper lobe opening was reduced in size and the mucous membrane appeared to be very hemorrhagic, but there was no evidence of any ulceration or fungating tissue visible at the orifice of the right upper lobe. The middle and lower lobe openings showed no gross pathology. The left side showed nothing more than just moderate inflammatory changes of the mucous membrane. By using a curved aspirator we were able to aspirate some material directly from the right upper lobe orifice and a moderate amount of purulent, blood-stained material was obtained, and this was sent to the laboratory for complete study, including cytologic, Tb smear and culture, yeast and bacteriological studies.

From the clinical course that this patient had been running and from the bronchoscopic findings, I was inclined to believe that this might be a primary inflammatory lesion, rather than a new growth, and I was particularly suspicious of a possible acid fast infection.

The studies of the aspirated material showed the Tb smear and culture to be negative. The study for yeast organisms was negative. The routine bacteriological culture showed abundant alpha streptococci; moderate Beta streptococci, both organisms sensitive to aureomycin and penicillin. The cytologic study was reported as suspicious for tumor cells.

The patient was discharged from the hospital on September 10, 1950, for therapy at home.

In spite of antibiotic therapy, he did not show any improvement, but instead became steadily worse, and was readmitted on September 21, 1950. During the period at home he had experienced sporadic daily elevations in temperature; weakness had become more progressive. The main reason for his readmission at this time was because of the marked swelling of his neck and face, which began five days previously. He now complained of a band-like constriction across the upper chest, with definite shortness of breath, which became very severe on the slightest exertion.

Physical examination showed—temperature 101; pulse 105; respirations 32; blood pressure 124/86. The positive findings centered about the face, neck and chest. In addition to swelling of his neck and face he had marked capillary venous dilations over the entire chest from the clavicles to the xyphoid region. No evidence of crepitus or subcutaneous emphysema could be palpated. The skin was full but showed no pitting edema. Both eyelids were moderately puffy.

Examination of the larynx revealed no evidence of edema. Both cords moved normally and the glottis was of normal size. There was no edema of the extremities at this time.

The first impression was the possibility of a penicillin reaction, so that antibiotic therapy was discontinued. He was skin tested with different antibiotics which were all negative.

On a regime of absolute rest, the swelling of the neck and face began to subside and although the dyspnea persisted, it was less severe. The patient also complained less of the tight constriction in the chest. However, in spite of this subjective improvement, he continued to maintain a septic temperature elevation curve.

The etiological factors which were considered and eliminated were:

Disseminated tuberculosis—sputa negative.

Mediastinitis—x-rays negative.

Septicemia—blood cultures negative.

Yeast Infection—no organisms found.

Trichinosis—skin test negative.

Brucella abortus—negative agglutination.

Hodgkin's disease—biopsy of axillary gland negative.

Bone marrow studies were negative for blood dyscrasia.

Pleural exudate was negative for tumor cells.

X-ray examination on September 26, 1950, was reported as follows by Dr. Paul Bishop:

"Re-examination of the chest, including a swallowing function, in an attempt to explain the marked increase in the mediastinal shadow on the right, also, when one compares the films taken at Abington Memorial Hospital, shows slight increase of the mediastinal shadow on the left.

After the completion of these studies, I believe this shadow is entirely vascular in origin. It is conceivable, with the patient's clinical history of what has occurred to him recently, that this could be engorgement of the superior vena cava, a superior vena cava thrombosis, and this lesion that we are seeing in the lung is not inflammatory but is a small pulmonary infarct. When following this shadow, that fits so well with the blood vessels of the hilar region and the mediastinum, it cannot be differentiated from the aortic shadow. The possibility of a dissecting aneurysm of the aorta must also be considered although the history certainly is not that of this latter entity. We are

also now able to appreciate the formation of small amounts of pleural fluid in both costo-diaphragmatic sulci; these were not present in our examination of four days ago.

Opinion: Rather marked change in the appearance of the great vessels and mediastinum on the right side over a period of one month's time, probably superior vena cava (Fig. 3).

Because of the persistent septic course the patient was placed on terramycin therapy, without any effect. During this period his dyspnea became severe and the patient began to show evidence of ankle edema, and beginning edema of both hands became evident. He frequently complained of the unbearable compressed feeling in his chest.

On the advice of Dr. John B. Flick exploratory mediastinotomy was performed on October 26, 1950.

"A curved incision 5 cms in length was made at the base of the neck about 1.5 cm above the clavicles. The incision was deepened to expose the suprasternal notch. A second incision was made over the middle of the sternum beginning at the level of the cervical incision and carried down to the tip of the ensiform cartilage. The sternum was divided in the median line with a Lebische knife. The incision was held open with a rib spreader. The tumor mass was not evident at once as the mediastinal structures were obscured by an enlarged thymus gland. This was removed. A hard, irregular mass approximately 8 cms. in diameter was noted to the right of the median line. This was adherent to the medial aspect of the right upper lobe, to the chest wall anteriorly over the right side of the mediastinum. The tumor had infiltrated the wall of the superior vena cava at its point of entrance into the atrium, the superior vena cava itself, and the left innominate vein."

The tissue obtained for histological examination was reported as epidermoid carcinoma, grade III.

Immediately postoperatively the patient showed definite improvement, but he subsequently redeveloped extensive edema of the extremities with circulatory failure and died on November 27, 1950.

CONCLUSION

Three cases of superior vena caval obstuction, caused by adjacent encroaching malignant lesions, are reported.

Although each one of these patients presented an individual diagnostic problem, all showed characteristic swelling of the neck and engorgement of the veins of the chest.

In one case the clinical course was very deceptive and in the early stages simulated possible antibiotic reaction and even Hodgkin's disease.

The endoscopic examinations did not reveal encroachment on the trachea or right main bronchus in any of these cases.

Cytologic examinations of secretions aspirated bronchoscopically were negative for tumor cells in two cases and suspicious in one.

Although vena caval obstruction was ultimately suspected in all the cases, the actual diagnosis was established by exploratory thoracotomy.

In all the cases it was impossible to resect the lesion.

255 South Seventeenth Street.

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DISCUSSION

Dr. Paul C. Samson (Oakland, Calif.): Over the years we have seen a number of patients with obstruction of the superior vena cava. Most of these have been due to malignant disease but obstruction can also develop due to pressure from mediastinal tumors and aneurysms as well as due to thrombosis from chronic infection. I emphasize this differential because vascular obstructions are commonly considered to be signs of inoperability in malignant disease, and while this is frequently the case it is not always true. Even in malignant disease, obstruction may be due to pressure and not to infiltration of the lumen. For instance, in malignant disease we have successfully resected a portion of the wall of the superior vena cava without obliterating the lumen and have relieved pressure by removal of benign tumors and by dissecting the vena cava free from aneurysms. Patients with thrombotic sequelae from infection form an infrequent but interesting group. I wish to record one case of this type which was subjected to operation. Much of the basic research on vena caval surgery has been reported by Frank Gerbode at Stanford University Hospital and on the basis of his work we were emboldened to attempt operative intervention. Five months before we saw this nineteen year old boy he became acutely ill with a high swinging fever, delirium, and signs of caval obstruction. The provisional diagnosis was acute lymphoma. X-ray and intensive antibiotic therapy were employed. The patient recovered but there was considerable disability due to the caval obstruction. This was manifested primarily by low-grade cyanosis of the head and neck, dizziness

when bending over, etc. The patient was explored through a median superior mediastinotomy. There were numerous caseous lymph nodes dissected but no active tuberculosis was found on subsequent culture. One cm of the superior vena cava and the distal portions of both innominate veins were thrombosed. This area was resected, the left innominate vein was closed permanently and the superior vena cava closed just above the azygos vein. An auto-graft of superficial femoral vein was used with end-to-end anastomosis above and side to side anastomosis below. The patient recovered. He lost his cyanosis, became asymptomatic and was able to return to work although his venous pressures remained elevated.

Should another case of this type present itself, my surgical approach would preferably be through a high postero-lateral right-sided transpleural thoracotomy. It might be possible in such a case, should the thrombosis be above the azygos vein, to use that vein for a portion of the graft.

LXIX

LARYNGEAL SEQUELAE OF ENDOTRACHEAL ANESTHESIA

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The endotracheal method of administering anesthesia has been improved considerably in the last decade, to the credit of the expanding group of physician anesthesiologists. This technic, through its reduction in mortality and morbidity, has made possible many advances in all forms of surgery, especially in that of the chest, head and neck. Its use is increasing in frequency by its spread to more locales and by its application to a greater variety of surgical cases.

This swing to endotracheal anesthesia is a welcome one—but—is the pendulum swinging too far? Is the anesthetist becoming so dependent upon an endotracheal tube that he is losing his finesse with other adequate forms of anesthesia? We ask this because we have encountered resistance, on occasions, in attempting to discourage the use of an endotracheal tube, especially in children, when other methods would have sufficed.

The advantages of endotracheal anesthesia are admitted but the disadvantages must be emphasized and re-emphasized to keep the anesthetist and surgeon from being lulled into a false sense of security.

Disadvantages, as listed by Adriani,[1] Gillespie,[2] and others, are:

1. The endotracheal tube is a foreign body with resultant irritation.

2. Lubricants may be irritating and in some instances may produce a local allergic reaction.

From the Division of Otolaryngology of the Stanford University Medical School, the Department of Otolaryngology of the Mt. Zion Hospital, and the Ear, Nose and Throat Division of the Letterman Army Hospital.

3. The lumen of the airway is lessened—especially important in children.

4. The patient must be under deeper anesthesia for intubation than sometimes is necessary for the surgical procedure.

5. There may be trauma to teeth, pharynx, nose or trachea during introduction. (This trauma we have witnessed too many times and agree with Moss[3] statement that "Ploughing up the pharynx with a laryngoscope in an attempt to dig out an epiglottis from a pool of blood and mucus is one of the least inspiring sights of modern anesthesia.")

6. Coughing or straining may cause increased venous pressure with undesired effects.

7. Bacterial flora from the nose may be carried to the lower respiratory tract.

8. Pulmonary sequelae can occur.

9. Laryngeal sequelae can occur.

It is with laryngeal sequelae that we are concerned in this presentation which includes the reports of eight cases of laryngeal obstruction with two deaths and seven cases of laryngeal granulomas.

The increasing number of laryngeal sequelae occurring in our own institutions suggested that some information might be gained from examining a number of patients immediately following endotracheal anesthesia.

Eighty consecutive patients who had had endotracheal anesthesia at the Stanford University Hospital were examined by indirect laryngoscopy on the day before surgery, post-operatively, and on the third to the fifth day. All were advised to report for further examination if symptoms developed after the fifth-day examination. The operative procedures varied from cholecystectomy, laminectomy, hysterectomy, orthopedic procedures, neck surgery, and a few adult tonsillectomies. Intubation was by both oral and nasal routes, with a variety of tubes, both with and without cuffs.

The anesthetists were either on the teaching staff or the resident staff of the hospital. The latter were supervised by instructors.

There were no immediate serious postoperative reactions in this group. In only two were preoperative abnormalities found. One had a small nodule on one vocal cord. This nodule was removed by direct laryngoscopy four days before intubation and was found to be

benign. The second had a left vocal cord paralysis (preoperatively) with no changes in the laryngeal findings postoperatively.

The 80 patients were all questioned as to hoarseness, sore throat, or other postoperative discomfort, and the larynxes were examined with a mirror.

All patients complained of a mild degree of throat discomfort during the first 24 hours. Three (excluding the tonsillectomized patients) complained of severe sore throat which persisted 48 to 72 hours.

Thirty-four complained of varying degrees of hoarseness. They were found to have a moderate degree of redness of the vocal cords and the arytenoid mucosa with some irregularity of the posterior third of the cords. In none did symptoms persist for more than five days. An additional 16 patients complained of hoarseness but the vocal cords and arytenoid mucosa appeared to be normal.

There was no hoarseness in 30 cases. Their larynxes were normal.

In 1948, Donnelly, et al[4] reported a similar study in 100 patients. The anesthetists recorded the intubation as traumatic in 38 of these, while mirror examination revealed objective findings of trauma in 60. Twenty-nine revealed vocal cord congestion, 10 membraneous glottic congestion, 2 mild edema of the epiglottis, 3 submucous puncture of the larynx, and the remainder indication of trauma to the palate, tonsil, or tongue. Wylie[5] reported on the findings on immediate direct examination of the larynx after removal of the tube in 100 cases. Forty-seven showed positive evidence of trauma about the pharynx, epiglottis, or glottis. The incidence was a little less in 50 cases in which the MacIntosh laryngoscope was used and curare added at the time of the induction.

It is apparent from our own studies, considering that laryngeal sequelae do occur, that 80 is an insufficient number of patients on which to base a conclusion. Actually, the survey tends to represent endotracheal anesthesia as being less formidable than it is because, although all of the 80 patients developed some type of a reaction to the endotracheal procedure, none developed any important sequelae. The study is further misleading in that the 80 patients included no children in whom the serious sequelae more frequently happen.

The cases to be presented demonstrate that serious sequelae do occur and that the endotracheal method of anesthesia is a formidable one. They show also that there are instances in which laryngeal or

vocal cord ulcerations that go on to abscess or granuloma formation may not be grossly recognized in the early stages (Cases 1, 2, and 8).

CASE REPORTS*

These cases of laryngeal sequelae from endotracheal anesthesia are divided into 2 groups: I. Laryngeal obstruction and II. Laryngeal granulomas.

I. Laryngeal obstruction

This series of 8 cases includes:

A. 4 patients, all children, with laryngeal edema, 2 of whom died. Tracheotomy was performed on 3 of this group but was not done on one of the patients who died.

B. 4 patients with occluding membraneous laryngitis and/or tracheitis.

TABLE I.

LARYNGEAL OBSTRUCTION FROM LARYNGEAL EDEMA

CASE	AGE	SURGERY	DURATION ENDOTRACHEAL ANESTHESIA	TRACHEOTOMY	RESULT
1. M. T. F.	6	Anastamosis right subclavian art. to pulmonary artery	3 hrs. 40 min.	Not done	Died 2nd day post-op.
2. D. L.	3	Ligation patent ductus arteriosus	3 hrs.	Done 2 hrs. after surgery	Died 4th day post-op.
3. J. D.	8	Ligation patent ductus arteriosus	?	Done 4 hrs. after surgery	Recovered
4. J. B.	7	Adeno-tonsillectomy-secondary	?	Done 28 hrs. after surgery	Recovered

CASE 1.—M. T. F., a Filipino girl, aged 7, with congenital heart disease. On February 25, 1949 she had an anastamosis of the right subclavian artery to the pulmonary artery. Endotracheal anesthesia

*We gratefully acknowledge the courtesy of Dr. Robert C. McNaught, Dr. Lewis F. Morrison, Dr. Lee Shahinian, and Col. Wendel A. Weller in permitting us to use cases of theirs which have been included in this report.

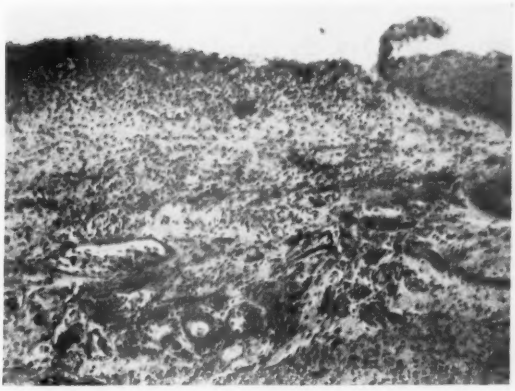

Fig. 1.—Superficial ulceration with fibrinous and fibrino-purulent exudate extending to margin of epithelium; extensive acute inflammation with edema in subepithelial tissues (100X).

Fig. 2.—Section through another area of the larynx of Case 1. Superficial ulceration with a thin layer of fibrin extending to margin of epithelium; severe edema with serum, fibrin, polymorphonuclear infiltration and dilatation of blood vessels (100X).

with a cuffless tube; duration: 3 hours, 45 minutes; the tube was easily introduced; cyclopropane, nitrous oxide and curare were used.

On February 26, 1949, the cyanosis which existed preoperatively showed definite improvement but the trachea and upper bronchi seemed to be full of secretion. The lungs were clear except for rhonchi, more marked on the left. The patient continued to show difficulty in breathing and at times became markedly dyspneic.

On February 27, 1949, at about 2:45 p. m., the respiratory distress was marked and the anesthesiologist was called to see the patient. He noted retraction of the suprasternal, intercostal and hypoxiphoid areas on inspiration with equal expansion bilaterally. His immediate impression was that of a partial obstruction high in the trachea or at the level of the cords. He applied topical anesthesia to the vocal cords. The patient's color and difficult breathing became worse so the anesthesiologist passed an endotracheal tube via the left nostril and stated that the obstruction appeared to be relieved. Artificial respiration (mouth to endotracheal tube) was done to assist in oxygenation but the patient soon expired (3:20 p. m.). The tube was immediately reinserted via the mouth but this procedure plus artificial respiration proved to be of no avail.

POST MORTEM FINDINGS*

Larynx: grossly there was moderate edema of the false vocal cords; the true vocal cords appeared normal. There was also some edema of the mucosa of the larynx. This edema had reduced the lumen to about half its size. There were no mucosal ulcerations. Microscopically large areas of mucosal sloughings were seen. They involved both the pseudo-stratified columnar and the stratified squamous portions in large strips and isolated patches. The denuded surfaces were covered with a thin layer of fibrin and polymorphs. Beneath the surface numerous polymorphs extended as deep as the mixed glands and striated muscles but not as far as the cartilaginous plates which were normal. Many of the superficial glands showed some necrotic elements due to the inflammation. There was also some edema of the tissues and marked congestion of the superficial vessels. Other regions of the mucosa were normal and showed well-developed submucosal lymph follices (Figs. 1 and 2).

The Trachea was of normal caliber throughout its length and the mucosal surface showed no ulcerations, hemorrhage or edema.

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*The pathology and microscopic sections in Cases 1 and 2 by Dr. Gerson R. Biskind, Chief of Department of Pathology of Mt. Zion Hospital.

Bronchi: The right and left bronchi and their branches revealed them to be patent throughout with no evidence of inspissated mucus or fresh secretions.

Lungs: The right and left lungs appeared normal in size and were not significantly retracted. The lobar fissures were developed. Section revealed normal crepitation and aeration throughout all lobes. Pressure expressed only a small amount of fluid. No local lesions were noted. The pleural cavities were clear except for a small amount of bloody fluid in the right. Microscopically there was a patchy congestion of the alveolar capillaries and very minimal atelectasis in some regions. The bronchi were normal and showed no inflammation.

COMMENT

It is obvious that the implications of a high tracheal or laryngeal obstruction should have been recognized much earlier and an otolaryngologist, not an anesthesiologist, should have been called to do a tracheotomy long before severe obstructive symptoms developed.

It is noteworthy in the post mortem report that, grossly, the ulcerations of the larynx were not identifiable but were very obvious under microscopic examination.

CASE 2.—D. L., a white girl, aged 3, with congenital heart disease (patent ductus arteriosus). On August 21, 1950, she underwent a ligation of a patent ductus arteriosus. Endotracheal anesthesia, intubated easily; duration: 3 hours; cyclopropane induction, nitrous oxide and curare.

Following surgery, after the endotracheal tube was removed, the patient became cyanotic, had thoracic retraction and breath sounds were absent on the left side. The endotracheal tube was reinserted and the trachea was aspirated. The cyanosis and retraction lessened and the breath sounds returned. The patient was returned to bed in fair condition.

Two hours later severe respiratory distress of the high obstructive type reappeared. A tracheotomy was done and the respiratory distress was immediately relieved.

The day following surgery the condition of the patient was only fair. The temperature rose to 102°. Respirations were rapid but not distressed. The temperature kept climbing and on the third postoperative day the trachea and bronchi were inspected with a bronchoscope inserted through the tracheotomy opening. There was

no crusting or any other evidence of tracheo-bronchial obstruction. The mucosa, however, was profusely injected throughout.

On the fourth post-operative day the patient died.

POST MORTEM FINDINGS

The larynx, grossly, was moderately edematous and its mucosa presented a profuse ecchymosis. (Figs. 3 and 4) Mucous and pus were present in the lumen. Ulcerated areas were present, denuded of epithelium. The denuded areas were covered with a thin layer of fibrin. Edema and inflammatory exudate were present in the submucosa. There was a distortion of the glands.

The lungs: The right lung weighed 80 grams and the left 90 grams. Both lower lobes presented a dusky appearance and seemed to be partly collapsed. Crepitation was markedly diminished in these parts. There were also areas of partial atelectasis scattered throughout the remainder of the lungs. The diagnosis at post mortem was:

1. Patent ductus arteriosus. Recent surgical ligation.
2. Extensive bronchitis and bronchiolitis with tenacious exudate.
3. Laryngeal edema. Tracheotomy.
4. Patchy atelectasis, primarily of both lower lobes.
5. Bronchopneumonia.

COMMENT

It seems from the report that the cause of death was extensive patchy atelectasis with secondary bronchopneumonia. One wonders if this might not have been due in part to too great relaxation as the result of the drugs used during the anesthesia in this very young child.

CASE 3.—J. D., a white girl, aged 8, with congenital heart disease (patent ductus arteriosus). On February 5, 1948, a ligation of patent ductus arteriosus was done. Endotracheal anesthesia.

As the patient was reacting from the anesthetic, she was having increasing respiratory difficulty of the high obstructive type. Examination of the larynx by the indirect method revealed marked redness and edema of the false and true cords. A tracheotomy was performed and the respiratory distress was immediately relieved.

The patient did well until two days later when respirations became difficult. A bronchoscope was passed into the trachea via the tracheotomy wound. The trachea and main-stem bronchi were lined

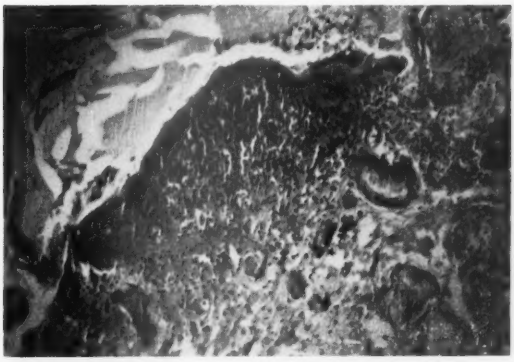

Fig. 3.—Section through larynx of Case 2. Lumen filled with mucus and pus; epithelial layer missing and replaced by a thin layer of fibrin; fibrino-purulent exudate in the submucosa; marked edema and distortion of glands (100X).

Fig. 4.—Section through another area of the larynx in Case 2 showing absence of epithelium and extensive subepithelial edema. This section was made four days after the intubation and indicates how persistent the edema may be in the larynx of a child (100X).

with heavy crusts which were removed by suction and forceps. The course from this point on was uneventful.

CASE 4.—J. B., a white boy, aged 7, with chronic nephritis and beginning deafness. The recurrent adenoid tissue was to be removed and endotracheal anesthesia was selected because it was thought that it might be the method of choice in the presence of chronic nephritis.

On January 27, 1948, his adenoids were removed. Endotracheal anesthesia.

About two hours after surgery the patient showed signs of upper respiratory obstruction. In the afternoon of the same day the obstruction seemed less. The patient's condition remained unchanged until the afternoon of the following day at which time respiratory difficulty of a high obstructive type again became marked. A tracheotomy was done and the respiratory distress was immediately relieved. It was felt that most of the patient's edema was in the subglottic area.

The tracheotomy tube was removed on February 16, 1948. The remaining course was uneventful.

COMMENT

It is the conviction of some of us that endotracheal anesthesia should not be used for an adeno-tonsillectomy in children.

CASE 5.—L. F., a white female, aged 59, who had a gastric resection June 10, 1949. Endotracheal anesthesia; difficult intubation in which several unsuccessful attempts were made until a relatively small No. 26 McGill tube was passed; duration: 3 hours, 45 minutes.

Signs of increasing laryngeal obstruction appeared during the first postoperative 24 hours and in 36 hours examination revealed the glottic and subglottic tissues to be covered with a fibrinous cast leaving a minimal chink. A tracheotomy was contemplated but was not necessary when it was found that the cast could be removed by suction and grasping forceps by indirect laryngoscopy. Laryngoscopy and bronchoscopy five days later revealed reddened vocal cords and reddened adjacent tracheal mucosa. Follow-up for six months revealed no further abnormalities.

CASE 6.—K. K., a white female, aged 12. On July 4, 1950, a spinal fusion was done. Endotracheal anesthesia, tube inserted without difficulty; Intracaine* ointment used; duration: 3½ hours.

*B-diethylamino ethyl p-ethoxy benzoate.

TABLE II.

B. LARYNGEAL OBSTRUCTION FROM OCCLUDING MEMBRANEOUS LARYNGITIS AND/OR TRACHEITIS.

CASE	AGE	SURGERY	DURATION ENDOTRACHEAL ANESTHESIA	FINDINGS	TREATMENT	RESULT
5. L. F.	59	Gastric resection	3 hr. 45 min. Intracaine oint. on tube	Membrane in larynx and trachea	36 hrs. post-op. removed by indirect laryngoscopy	No recurrence
6. K. K.	12	Spinal fusion	Intracaine oint. used. Duration: ?	Membrane in larynx	28 hrs. post-op. removed by direct laryngoscopy	No recurrence
7. H. S.	55	Hysterectomy	1 hr. 30 min. Nupercaine oint on tube	Membrane in larynx	48 hrs. post-op. removed by indirect laryngoscopy	No recurrence
8. R. G.	42	For common duct obstr.	2 hr. 45 min. Intracaine oint. on tube	Membrane between and below vocal cords	56 hrs. post-op. removed by direct laryngoscopy	Recurrence in 36 hrs. Again removed

Apparently the patient had developed increasing dyspnea following the removal of the endotracheal tube, and at the end of 28 hours an otolaryngologist was called in. At that time the patient was semiresponsive, gray, and displayed marked indrawing of the suprasternal notch and intercostal spaces. A tracheotomy was planned and a laryngoscope was introduced without anesthesia for the insertion of a Portex tube prior to tracheotomy. Exposure of the glottis revealed that its lumen was occupied by a fibrinous membrane leaving a tubular airway of only ⅛ inch (3 mm) in diameter. The membrane was dislodged with the tracheal tube and was caught with the metal suction tube. It was possible to remove this entire membrane with strong suction through the metal tube. There was instant relief with no further recurrence of the respiratory distress.

CASE 7.—Mrs. H. S., a white woman, aged 55. On June 12, 1948, a hysterectomy was performed. Endotracheal anesthesia; tube inserted without difficulty; Nupercaine** ointment used; duration: 1½ hours.

The otolaryngologist was called 48 hours postoperatively because of increasing dyspnea and stridor.

Mirror examination of the larynx showed the false and true cords to be covered with a gray-yellow membrane leaving only a small space at the posterior commissure.

The larynx was anesthetized with 10% cocaine. Indirect laryngoscopy was done and the membrane grasped with a curved laryngeal forceps and removed. The specimen was very tough and was a complete cast of the interior of the larynx.

There was no recurrence.

CASE 8.—R. G., a Spanish-American woman, aged 42. On August 4, 1947, an exploration of obstruction of the common bile duct was done. Endotracheal anesthesia; duration: 2 hours 45 minutes; Intracaine was used on the tube. On the first postoperative day it was noted that the patient's voice was feeble and hoarse. About 46 hours postoperatively some difficulty in breathing developed and five hours later she developed an inspiratory stridor and was unable to phonate audibly.

In another three hours a mirror examination of the larynx was done. This revealed a grayish-white membrane projecting into the tracheal lumen just at the level, or below the level, of the vocal

**Butyloxycinchoninic acid diethylene diamide hydrochloride.

cords. The laryngeal end of this membrane was loose and had a ball-valve obstructing action on the larynx during respiration.

A direct laryngoscopy was done. The membrane was seen to be in the trachea just below the level of the vocal cords. It was removed as a cast in one piece. There was no bleeding from the underlying tissues after its removal. The vocal cords, as well as the laryngeal mucosa, seemed smooth and uninvolved. A bronchoscope was passed. The trachea and both main bronchi showed no abnormality other than redness. The respiratory distress was completely relieved when the fibrinous membrane was removed.

Two days later the patient had a coughing spell and suddenly developed respiratory distress, both inspiratory and expiratory, but mainly inspiratory. Mirror examination of the larynx revealed a recurrence of this gray-white membrane in the trachea adjacent to the larynx. This was removed by direct laryngoscopy. Bronchoscope was passed and smaller membranes were present in both main bronchi just below the carina. These were removed by the aspiration. Obstruction to breathing was completely relieved.

The same day and the following day the patient had noticed respiratory distress and she could not speak with an audible voice.

Two days later mirror laryngoscopy revealed that the whitish membranes were again reforming below the vocal cords but there was still a good airway. On the following day, however, another examination disclosed that the membranes had completely disappeared. There was no recurrence of respiratory difficulty from this time on.

Smears and cultures of the membrane removed at the first laryngoscopy revealed gram positive cocci in pairs and short chains, like pneumococcus, and gram negative rods, biochemically like proteus vulgaris.

Two weeks after surgery a skin patch test with Intracaine ointment (which had been used on the endotracheal tube) was done. This showed no reaction.

Five weeks postoperatively the patient developed hoarseness for which she did not seek attention until eight weeks postoperatively. Examination of the larynx revealed granulomas on both cords which will be discussed in further detail with the series on granulomas.

COMMENT

Cases 1 to 4 of laryngeal edema in children are grim reminders that endotracheal anesthesia cannot be used with abandon in chil-

dren. Holinger and Johnston[6] have pointed out that the glottic chink of the larynx of the new-born infant has an area of 14 sq mm. An edema of 1 mm of the mucosal surfaces will reduce this area to 5 sq mm or only 35% of the original area. By comparison with an equal mucosal change in the larynx of an adult, 1 mm of edema of all surfaces would not produce respiratory change or obstruction; it would manifest itself only by hoarseness.

The size, the composition of, and the skill with which the tube is inserted, can be controlled by the anesthetist, but the amount of motion or friction of the tube against the vocal cords during the anesthesia cannot. There is no way of predicting which patient will have postoperative laryngeal difficulty unless, of course, the anesthetist knows that, in a given case, unusual trauma has taken place.

It is difficult for us to understand why, in children, endotracheal anesthesia should be used for an adeno-tonsillectomy in any instance. Anesthetists have argued that they protect the patient's airway from the blood overflow that occurs in the hands of certain surgeons. This may be so in some instances but it does not seem reasonable that an endotracheal tube should be used as a substitute for good surgery.

Why an occasional patient after endotracheal anesthesia should develop an occluding membrane within the larynx is not known. It has been postulated that this may be a local allergic response to the material of the tube itself, to the solution in which it has been soaked or to the lubricant on the tube. Case 8, one of the series with an occluding membrane, was patch-tested with the lubricant (Intracaine). The reaction was negative. A test such as this in a single instance proves nothing except that further investigation is in order.

Paluel J. Flagg,[7] in a recent survey, received responses from 99 otolaryngologists stating that they had, at one time or another, been called for consultation in postoperative trauma following endotracheal intubation. With Dr. Flagg's permission, I report some of the statistics resulting from this questionnaire.

Thirty-one of these otolaryngologists describe specific injuries as: hemorrhage of the vocal cords, 2; irritation from the tubes soaked in Benzalkonium (zephiran); injury to the mucosa of the trachea, 3; laceration of the vocal cords, 5; edema of the vocal cords, 5; hoarseness; mucosa scraped from the cords; trauma to the carina; paralysis of the vocal cords; edema of the trachea, 2; perforation of the trachea, 2; laryngeal edema; tracheal webs, 3; leucocytic membrane of the cord and trachea; traumatic laryngitis.

Of those called in as consultants, 42 reported performing tracheotomies for reaction to intubation; 10 stated they knew of deaths in which intubation was a factor.

Ranson[8] reported a fatal case of tracheitis and laryngeal obstruction after uneventful intubation and anesthesia and surmised that the reaction was due to the agent (biniodide of mercury) used for sterilization of the tube. Shaw[9] reported a case of acute laryngeal edema necessitating tracheotomy in a patient under prolonged endotracheal anesthesia after a difficult intubation. The tube had been lubricated with petrolatum.

Flagg[10] reported marked laryngeal spasm post-operatively in a 2-year-old child following intubation but the reaction subsided in 2 hours.

Wylie[5] reported a death following endotracheal anesthesia in which there was prolonged laryngeal spasm during the attempt at intubation. When the tube was finally passed, blood-tinged fluid was sucked from the tube. On completion of the surgery and after the tube was removed, laryngeal spasm recurred and, although another tube was inserted, death occurred. Death was due to pulmonary edema. Wylie felt that with present-day relaxing drugs available, spasm should not be a factor, although he concedes that too much relaxation may cause respiratory depression.

Donnelly[4] reported post-operative laryngeal edema in 2 children intubated with large tubes; obstructing symptoms persisted 4 hours. He reported a death from esophageal perforation during a difficult intubation in which a stylet was used.

Crispell and Hampton,[11] reviewing endotracheal anesthesia in children, reported 2 tracheotomies from laryngeal edema in 337 intubations.

II. LARYNGEAL GRANULOMAS

CASE 8.—R. G., a Sanish-American woman, aged 42, previously mentioned under the group of cases having laryngeal obstruction from an occluding membraneous laryngitis following an endotracheal anesthesia lasting 2¾ hours. She developed hoarseness three weeks postoperatively but did not seek attention until eight weeks postoperatively. Mirror examination of the larynx revealed large, smooth masses semi-pedunculated, on the vocal cords at the vocal processes. The masses were pale and, grossly, appeared more like polyps than granulomas. Direct laryngoscopy was done on October

Fig. 5.—Granuloma of vocal cord of Case 8 eight weeks after intubation. Epithelium absent from the surface which is covered by a smooth layer of fibrin undergoing organization (30X).

Fig. 6.—Granuloma of vocal cord of Case 8. Surface covered by smooth layer of fibrin extending to epithelium. Organization of exudate with capillaries, fibroblasts and chronic inflammatory cells (100X).

2, 1947, and the masses were removed. Microscopic sections of these are shown in figures 5 and 6.

Follow-up examination in November 1947, revealed the left vocal cord to be smooth but there was a recurrence of a small granuloma over the right vocal process. This was examined from time to time and by April, 1948, the recurrent granulation tissue had disappeared spontaneously and both vocal cords were smooth. The patient's voice was clear.

CASE 9.—This case is interesting because the patient had surgery three times within a thirteen month period, and after each operation she developed vocal cord granulomas.

Mrs. E. R., had surgery on December 31, 1948, for a ruptured nucleus pulposus. Endotracheal anesthesia with sodium pentothal, curare, nitrous oxide and ether; duration: 3 hours 15 minutes. Four days later it was noted that the patient was hoarse. Two and one-half months later, on March 17, 1949, the patient was examined because of hoarseness. Mirror examination of the larynx showed a granuloma, approximately 5 mm in diameter, somewhat pedunculated, and attached to the left vocal cord at, and slightly anterior to, the vocal process. There was on the right vocal cord a small, shallow, ulcerated area in a similar position without evidence of granulation tissue formation. On April 2, 1949, the granuloma was removed from the left cord by the indirect method under local anesthesia. It is of interest that approximately 13 days following this procedure the patient, while under severe fright and strain, screamed several times and immediately became quite hoarse. Examination of her larynx on April 22, 1949, showed the entire right cord was covered with a large hematoma. There was no evidence of surface ulceration on either cord. This hematoma regressed and by May 12, 1949 both cords were found to be normal.

Eleven months later, on November 14, 1949; further surgery for the nucleus pulposus was done. Endotracheal anesthesia; duration: 2 hours 15 minutes. Two days later examination of the larynx revealed granulomas on both cords at the vocal processes. The one on the right regressed rapidly. At some time between November 4th and December 19th, 1949, the patient coughed and spat out a small piece of tissue which she described as being the size of a split pea. Examination of the larynx on December 20, 1949, showed no recurrence of the granulomas.

On December 21, 1949, a laminectomy was done. Endotracheal anesthesia: duration, 4 hours. Thirteen days later examination of

TABLE III.

LARYNGEAL GRANULOMAS.

CASE	AGE	SURGERY	DURATION ENDOTRACHEAL ANESTHESIA	ONSET HOARSENESS	VOCAL CORDS INVOLVED & WHEN NOTED	TREATMENT	RESULT
8. R. G.	42	For common duct obstr.	2 hr. 45 min.	5 wks. post-op.	8 wks. post-op. Both cords	Removal direct laryngoscopy	Recurrence but resolved without further treatment
9. E. R.	adult	1. Ruptured nucleus pulposus	3 hr. 15 min.	4 days post-op.	10 wks. post-op. lt. cord	Removed 12 wks. post-op. indirect method	1. See 2
		2. Re-op 11 mo. later	2 hrs. 10 min.	Shortly post-op.	10 days post-op. Both v. cords	Spontaneous ejection	2. See 3
		3. Re-op in 5 wks. Laminectomy	4 hrs.		2 wks. post-op. rt. cord 4½ wks. post-op. left cord	Left one 4 wks. post-op.	3. No recurrence
10. A. F. D.	49	Fractured ankle	?	2 wks. post-op.	6 wks. post-op. both cords	Removed 1) Direct Laryngos. 2) Indirect Laryngos. 3) Direct Laryngos.	3 recurrences Recovery in one year

11.	M. L.	44	Gastric resection	3 hrs. 30 min.	2 wks. post-op.	3 wks. post-op. right cord	None	Recovery after 4 mo. coughed up granuloma
12.	M. F. P.	48	Gastric resection	?	4 mo. post-op.	6 mo. post-op. left cord	Removed direct laryng.	No recurrence
13.	B. P. S.	39	For herniated nucleus pulposus	?	Since surgery	5 wks. post-op. both cords	Refused treatment	Vocal cords normal 5 mo. later
14.	M. C.	Early 30's	Thyroidectomy	?	4 mo. post-op.	4 mo. left cord	Spontaneous ejection	No recurrence

the larynx showed a small granuloma on the right vocal cord and a small superficial ulcer over the left vocal process. Re-examination in another 18 days showed improvement of the appearance of both vocal cords. There was a persistent granuloma on the left vocal cord at the site of the previous granulomas. This was removed by the indirect method.

Re-examination of this patient at various intervals, and last on March 9, 1951, showed the larynx to be normal in all respects.

CASE 10.—Mrs. A. F. D., a white woman, aged 48. On October 3, 1949, there was a surgical reduction of a fracture of the ankle. Endotracheal anesthesia. Patient had a delayed penicillin reaction with swollen hands, mouth, and lips two weeks following surgery, after which hoarseness was noted. Hoarseness progressed and on December 15, 1949, examination revealed granulomas at the vocal processes of both vocal cords. These were removed on December 22, 1949, by direct laryngoscopy. Hoarseness and the granulomas recurred. The granulomas were removed on April 14, 1950, by indirect laryngoscopy. About the middle of May examination showed further recurrence and on June 15 these were removed by direct laryngoscopy and the bases were dessiccated. It is to be noted that the recurrence on the right vocal cord at this time was not at the vocal process but about the middle of the vocal cord. On September 6, 1950 the voice was very clear but examination showed a small nubbin at the base of the left vocal cord near the vocal process. This was let alone and the patient was seen again on December 6, 1950. Both vocal cords appeared smooth and clean.

CASE 11.—M. L., a white female, aged 44. A gastric resection was done February 25, 1947. Endotracheal anesthesia; duration: 3½ hours; Intracaine ointment was used on the tube. There was mild discomfort in the throat postoperatively. There was some mild hoarseness after dismissal from the hospital. Examination three weeks postoperatively showed the presence of a nodular mass, which appeared to be a granuloma, on the posterior third of the right vocal cord. This patient did not return for immediate follow-up therapy as requested. She remained hoarse for 4 months. This hoarseness disappeared very suddenly one morning when she awakened. It is postulated that the granuloma was spontaneously dislodged and coughed up. Subsequent laryngeal examination revealed normal vocal cords.

CASE 12.—Mrs. M. F. P., a white woman, aged 48 years. A partial gastrectomy was done June 10, 1950. Endotracheal anes-

Fig. 7.—Granulomas of vocal cords, Case 12.

thesia. Hoarseness developed in October, 1950. The patient appeared for examination on December 12, 1950. Examination of the larynx showed a granuloma projecting from the mid-portion of the left vocal process. It was large and flipped up and down. When it moved upwards, it prevented the cords from approximating. There was also a small granuloma near the tip of the right vocal process. Direct laryngoscopy was done on December 14, 1950, and the granulomas were removed. The larger one on the right side measured 8 mm by 10 mm. The postoperative course was uneventful. There were no recurrences (Fig. 7).

Case 13.—B. P. S., male, aged 39 years. On May 14, 1947; he was operated upon for herniated nucleus pulposus. Endotracheal anesthesia. He reported for examination on June 23, 1947, stating that he had had some painful swallowing and hoarseness since the operation. Examination showed granulomas at the vocal processes of both vocal cords. Removal of these by direct laryngoscopy was suggested but the patient refused operation. He was re-checked on October 22, 1947, at which time his hoarseness was gone. Mirror examination of the larynx showed normal appearing vocal cords.

Case 14.—M. C., a male in his early thirties. Thyroidectomy was done March, 1947. Endotracheal anesthesia. About four months later he had some difficulty in talking. Examination of the larynx revealed a flipping, pedunculated granuloma near the tip of the vocal process on the left vocal cord. The night before the scheduled removal the patient attended the theater. He laughed heartily during a vaudeville act and experienced a stinging sensation but continued to enjoy the show. About 5:00 a. m. the patient awakened, cough-

ing, and felt a "grape" in his mouth. He came in next morning and presented the specimen which was an ovoid granuloma approximately 7½ mm in its greatest diameter with a hemorrhagic stalk. Examination of the larynx showed a very smooth vocal cord. There was no recurrence.

TABLE IV.

SUMMARY OF CERTAIN FEATURES OF EACH GROUP.

I. Laryngeal Obstruction	
	All were children; 3 females 1 male
A. From laryngeal edema 4 cases	All had evidence of some respiratory difficulty shortly following surgery
B. From occluding membraneous laryngitis and tracheitis 4 cases	All were females Serious respiratory difficulty developed after 24 hours post-op.
II. Laryngeal granulomas—7 cases	
1. Sex: 5 females; 2 males	
2. Age: 40-50 predominant	
3. Involvement: 4 both v. cords 3 one v. cord	
4. Treatment: 4 removal 3 spontaneous ejection	
5. Result: Complete recovery in all cases	

COMMENT

Reports of laryngeal granulomas following endotracheal anesthesia are becoming relatively common.

Flagg,[7] in his survey previously referred to, received replies from 34 surgeons reporting at least 101 cases of granuloma. These tumors were noted from four weeks to four months after intubation. The literature contains 41 case reports of post-intubation granulomata with at least 10 to 15 further cases mentioned in discussions of papers read at various society meetings. New and Devine[12] noted that of 33 cases of patients with contact ulcer granuloma, 9 gave a history of recent intubation.

The most common site was near the vocal process of the arytenoid and, in the majority, the lesions were bilateral.

Initially, there is an ulceration of the epithelium of the vocal cords. This ulceration is probably minute and may not be recognized even on careful examination directly after the completion of the intubation. The development of a thin layer of fibrin is an additional factor which makes it difficult to recognize the presence of an ulceration. In Case 8, when the vocal cords were carefully examined following the removal of the fibrinous membrane, there was nothing to suggest an ulcer on either vocal cord, and yet this patient later developed granulomas of both vocal cords. It seems likely that the presence of fibrin covering the ulcer, having the same color as the vocal cords, made it difficult to recognize any abnormality at that time.

It is probable that in most cases the granulomas would disappear without surgical intervention. The ingrowing cordal epithelium gradually chokes off the stem of the granulation tissue until it finally drops off and is coughed up. For this reason Kearney[19] and also McLaurin[21] suggested delaying the removal of the granuloma until it becomes pedunculated, to cut down the number of recurrences. We concur in this, excepting in those cases in which the granulomas may be so large as to severely impair respiration or phonation.

It seems that all patients get well but in an occasional one convalescence may be prolonged. This was true in Case 10 in which the patient required a full year for complete recovery.

CONCLUSION

Each time we have tried to discuss calmly with an anesthetist the problems of endotracheal anesthesia, especially in children, we have been deluged with an avalanche of statistics showing that, in certain institutions, intubations of both adults and children run into the thousands without serious sequelae.[29] We accept these experiences and concede that these anesthetists have reached a perfection in technic that is reflected in so few complications.

Rather than be influenced by these most favorable statistics, however, we should be guided by facts within our own experiences. The 15 cases of laryngeal sequelae herein reported were collected from five different hospitals with which we are affiliated and very likely are a representative cross-section of the experiences in similar communities. They reveal that important laryngeal sequelae can occur from endotracheal anesthesia in both adults and children and that they are not uncommon in children.

It is felt that endotracheal anesthesia should not supplant other adequate methods of anesthesia in children.

SUMMARY

1. The results of a study of 80 consecutive patients who had had endotracheal anesthesia are given.

2. Cases of laryngeal sequelae following endotracheal anesthesia are reported. These are:

I. Eight cases of laryngeal obstruction
   a. from laryngeal edema (4 cases)
   b. from occluding membranous laryngitis or tracheitis or both (4 cases)

II. Seven cases of laryngeal granuloma.

3. The opinion is expressed that endotracheal anesthesia should not supplant other adequate methods of anesthesia in children.

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DISCUSSION

PALUEL J. FLAGG, M.D. (New York City): I should like to compliment Dr. Baron for his excellent presentation. He has brought to a focus, through the recitation of a specific group of cases one of the findings of my survey on "The Incidence and Control of Trauma accompanying Endotracheal Anaesthesia" which appears in the Archives of Otolaryngology for April of this year.

Our views regarding the best method of eliminating this pathological reaction differ. Dr. Baron feels that endotracheal anaesthesia should be used less frequently, that some other form of general anaesthesia should be used. My impression is that there is no substitute for this technique but that the details of its common usage may well be improved. In other words instead of eliminating the technique, improve its use.

Let us consider the following: The shadow of impending death from aspiration and from asphyxia falls over all surgery of the head and neck performed under general anaesthesia. This shadow—this hazard, is eliminated by the endotracheal tube. In the present state of our knowledge the use of the endotracheal tube for this type of surgery is mandatory. There is no other mechanical means of protection available. Indeed a failure to use this protection in the case of accidental death from asphyxia or from aspiration may shortly be regarded as malpractice.

As Dr. Baron has clearly pointed out however, the use of the endotracheal tube has been followed by grave pathological reactions. It has also been shown that such reactions are most unusual in the practice of Bronchoscopy, the reported incidence being 2% (see Survey refered to).

The problem therefore is to translate if possible, the routine technique of bronchoscopy, suitable exposure, relaxation and tube sizes (MM in Outside diameter) into anaesthetic technique.

This field is *not* obtainable by barbiturates except supported by curare and topical Cocaine or Pontacaine. This field *is* obtainable with ether anaesthesia and has paralleled that of bronchoscopy for the last twenty five years in this speakers hands. It is humiliating to retrace our steps in the use of anaesthetic agents but the success to be achieved justifies this course. The reward is great-the elimination of asphyxia, protection against aspiration and freedom from post operative pathology. There is but one difficulty—one must learn how to administer ether.

SHIRLEY H. BARON, M.D. (closing): I should like to thank Dr. Flagg for his kindness in coming up this afternoon and discussing this paper. I believe that if our cases had been done with his type of technique, skill and caution, I would not have spoken so strongly against routine intubation for certain conditions. I know that he is right about medication because we have had the problem of overmedication and over-premedication.

I enjoyed seeing Dr. Friedberg's slides; they are very good slides.

LXX

BRONCHOGRAPHY IN THE INFANT AND VERY YOUNG CHILD

FERNAND G. EEMAN, M.D.

(Guest Speaker)

GHENT, BELGIUM

Bronchography, as is often the case with new steps in medicine, seems to be the consequence of a lucky mishap. Weingartner,[1] in a communication presented in 1914, relates the accidental penetration of bismuth into the tracheo-bronchial tree during the radiological examination of a patient suffering from a carcinoma of the esophagus. His paper is probably the first scientific account of a bronchography.

Although accidental this makes bronchography a procedure more than 35 years old. The innocuity of such a happening led to the consideration of the possibility of using it as a new technique of lung examination. The first searchers foresaw its remarkable diagnostic qualities. Among those must be mentioned Chevalier Jackson[2] whose pioneer work involving his earlier experiments with bismuth powder was published in 1918.

Two years later appeared the first publications of Lynah[3, 4] on the same subject. That author also used bismuth, not as powder but in a mixture.

Sicard and Forestier[5] in a publication in 1922, were the first to advocate the use of lipiodol as a contrast medium. They were followed by many others. The experimental and clinical research had been performed by Forestier and Leroux[6, 7, 8] who made their first satisfactory bronchographies towards the end of 1921.

Enormous interest was awakened by the work of these authors and during the following years a tremendous number of publications came to light. At first these were mostly concerned with technique but progressively the diagnostic value of bronchography was recognized, discussed and stressed.

To show how rapid the progress of the new technique has been, let us remember that at a symposium on lipiodol held in 1926,

Pritchard[14] reported the result of his experience based on a series of a thousand consecutive bronchographies. For a quarter of a century, bronchography has been a recognized diagnostic procedure, commonly used everywhere.

From the beginning, some authors have been attracted by the problem of bronchography in the child. Chief among them must be quoted the name of Sergent[15] who published a paper on the subject in 1924. A few months previously Armand-Delille,[16] in a communication before the Société Médicale des Hopitaux de Paris studied this particular aspect of bronchography. His paper is probably the first devoted to the subject of bronchography in young patients.

The flow of communications on the subject of bronchography has not decreased during the last two decades but one is struck by the negligible number of them devoted to young patients and the almost total absence of research made on young children and still more on infants.

Although bronchology has made a tremendous progress in Europe it is interesting to note that Rap and Smelt in their remarkable book on the bronchial tree published in 1947 in the Dutch language[17] devote less than two pages, mostly technical, to the subject of bronchography in children.

They do not seem to consider it possible to do bronchographies in children under the age of 4 or 5 years. Even in older children they judge that a unilateral bronchography cannot be performed. Bronchography in infants and very young children is not even mentioned. Nevertheless the authors agree on the great usefulness it could have, taking into consideration the brilliant results of lung surgery in children and its future possibilities.

The same remark applies to the splendid and beautifully illustrated treatise of Huizinga and Smelt[18] published in 1949 and devoted entirely to bronchography. Out of 270 pages, only one is devoted to bronchography in children. It mentions the possibility of performing bronchographies in children under the age of 8 to 9 years by using the transglottic catheter introduced by means of direct laryngoscopy, without anesthesia. The authors give some useful information on the technique but do not lay much stress on its results. This, together with the absence of published bronchograms in children tends to show that it has been tried on a moderate scale only. The authors are able to assert nevertheless that they have never seen any injurious results following lipiodol examination in children when correctly performed. They rightly consider that this is probably chiefly due to the fact that, as no anesthesia is used,

all the lipiodol is quickly expectorated. The recent book of Soulas and Mounier-Kuhn[19] also published in 1949, forms another important and original contribution to Bronchology, yet only one of its 650 pages is devoted to bronchography in children. The few hints on technique may be summarized as follows:

a) transglottic injection with a syringe in children from 10 to 15 years,

b) injection through a nasal catheter in children from 4 to 10 years,

c) injection through the bronchoscope in children from 3 to 4 years, under general anesthesia.

This last shows that the authors consider bronchography as a rarely to be performed examination in children of that age, justifying such extreme measures as general anesthesia. They further prove it by stipulating that bronchography shall only be performed when the indications are imperative as for instance the need for exact localization of bronchiectasis before surgery. Under the age of three years, bronchography is not even considered by these authors. This tendency to exclude young children from the diagnostic benefit of bronchography still prevails.

A change of attitude might have been expected, following the article of J. B. Miller, F. Mann and H. H. Abramson[22] on topical anesthesia by nebulization, published in 1949. It mentions the possibility of applying this technique to children under 4 years of age with satisfactory results and contains the actual bronchogram of a 5-year old child.

The advisability of these examinations in children is again discussed in a technical paper by J. B. Miller, William H. Conyers and Norman Dinhoffer[20] published in June 1950 in which the authors assert there is no need to avoid it or to fear its consequences. Nevertheless the authors do not mention any age limit and are seemingly not concerned with young children. The positioning technique, described in the article and the illustrations applies only to older children whose cooperation could be expected.

Why is it that bronchography is seldom performed in children under 7 or 8 years of age and is practically never considered in younger children or infants?

The answer is obvious. Everyone agrees with Miller, Conyers and Dinhoffer who open their article with this sentence: "Bronchography is performed in far fewer cases than is desirable." There

must be reasons explaining this unquestionable fact and it might be worth while to examine them and estimate their value.

The first reason which shall be put forward is the danger of this procedure in young children. This assertion may not be true and deserves further examination. One may sum up the various dangers of bronchography as follows:

a) the risk of the anesthesia.

b) the risk of the technical procedure of introduction of the opaque oil.

c) the risk of respiratory trouble due to the filling of the tracheo-bronchial tree.

d) the risk of intoxication due to iodine.

These risks undoubtedly exist but they are relatively remote and have not deterred the endoscopists from applying this diagnostic investigation to adults and adolescents with very useful results. Are these risks actually greater in infants and young children and, if so, are they sufficient to discard the use of bronchography in young patients?

Let us examine each of these risks separately.

a) The risk of anesthesia.

The actual tendency is to replace the more toxic products by less toxic ones. That is why cocaine has been gradually abandoned in favor of synthetic products such as pontocaine, xylocaine and others whose greater activity permits a lower dosage and whose toxicity is limited. Another tendency is to use a lesser quantity of these drugs combined with a better technique of application: either a very fine spray as advocated by Carabelli[21] or even nebulization as suggested by Miller, Mann and Abramson.[22] It is a great advantage to administer the anesthesia slowly, allowing plenty of time for the induction. It is now generally recognized that it takes from 20 to 30 minutes to obtain a satisfactory anesthesia without resorting to overdosage for quick results. The application of these principles to the anesthesia in young children as in adults, diminishes the risk to a trifling degree.

Are children actually more sensitive to local anesthetics than adults? This certainly has not been proven. I look upon this theory as a mere assumption accepted without much control and my personal experience makes me suspect that it might even be the reverse. We may thus conclude that the fear of an increased risk due to an-

esthesia is insufficient to deter us from using it in young children. The real difficulty lies in its application in the very young.

Is anesthesia always a necessity in order to perform a satisfactory bronchography in the young child? In the infant and the young child the transglottic catheter or the bronchoscopic methods will often permit one to obtain technically sufficient bronchograms without resorting to anesthesia. This nevertheless requires quick and well-timed teamwork from the endoscopist and the radiologist. The fear of a greatly increased risk in young children due to anesthesia may thus be discarded. The possibility and even advisability of doing without any anesthesia in many cases does away with this risk altogether.

b) The risk of the technical procedure of introduction of the opaque oil.

Is this risk greater than in adults?

The answer to this question depends on the procedure itself. One may assume that the introduction of a rigid instrument will be more traumatizing for the delicate laryngeal tissues and bronchial mucosa or skeleton of a very young child especially if the procedure involves bodily mobilization of the patient. Without perfect teamwork this will undoubtedly be true. With careful and intelligent co-operation between the endoscopist and his assistants experience tends to show[23] that this is not the case. It seems nevertheless advisable to resort to bronchoscopy only when other techniques cannot be applied or are inadequate. The same rule applies to the transglottic rigid catheter method. Nevertheless, an experienced endoscopist must not hesitate to use one of these techniques whenever indicated.

The soft catheter introduced either through the nose or introduced directly into the larynx through the mouth is just as inoffensive in children as in adults. The same applies to the simplified per-nasal method devised by Hicguet and Hennebert of Brussels and largely used by Forestier and Leroux.[24]

In many instances this method will give unexpectedly gratifying results with no technical risk at all. Here again we may conclude that the fear of accidents due to a traumatizing technique is over-estimated and is very limited in trained hands.

c) The risk of respiratory trouble due to the filling of the tracheo-bronchial tree. We must admit that the risk exists in children quite as much as in adults but not more. Over-filling alone is dangerous. If one keeps in mind the very much smaller capacity

of the tracheo-bronchial tree in the very young and injects only the reduced amount often sufficient to delineate it, this danger of respiratory trouble due to over-filling will not be greater than in the adult. This amount will vary greatly according to the patient. In an infant, 1½ or 2 cc will often be sufficient whereas in some young bronchiectatic patients very much larger amounts of iodized oil may have to be introduced, without danger of over-filling. This is another of the many good reasons why the instillation of the radio opaque oil must be done under fluoroscopic control. The danger of respiratory trouble depends thus largely on the ability of the endoscopist, his professional knowledge and his carefulness. These qualities must be used to their full extent when applying delicate and precise techniques in young children.

d) The risk of intoxication due to iodine.

This risk seems to have been overestimatd in adults and there is no reason why it should necessarily be greater in young children. This danger of intoxication has been very much feared by the first experimentors and incited them to great prudence.

Nevertheless, in 1928 Sicard and Forestier, reviewing the already abundant literature on the use of lipiodol[25] stated that general manifestations of iodism were very rare, always slight and of short duration. Local accidents with the exception of an eventual irritative cough during a few hours in some patients, are practically nonexistent. To diminish them still further, a few precautions should be taken:

1) abstain from the use of iodine suspensions in known cases of acute tuberculosis except when the examination is urgently indicated.

2) explore each individual patient's sensitivity to iodine by skin-tests two days before a proposed bronchography.

3) inject the necessary amount of lipiodol but not more.

4) abstain from general anesthesia which slows down the spontaneous expulsion of the oil suspension through coughing and expectoration.

5) quicken this natural expulsion, either by immediate aspiration, easily performed when the endoscopic technique is used or by postural drainage in the various but inversed positions used for segmentary bronchography. To be effective this procedure should last from 20 to 30 minutes.

The absence of any kind of anesthesia is often advisable in very young patients as it favors the quick expulsion of the iodine suspension and diminishes in the same proportion the toxic risk.

After 30 years of experimentations, most recent authors come to the conclusion that the iodine suspensions generally used are devoid of any real toxicity, if not entirely innocuous. In their very conscientious book, Huizinga and Smelt, after careful study of the literature, consider the danger of intoxication as negligible: "Summing up it may be stated that the introduction of lipiodol in the air passages only very rarely causes acute or chronic harmful effects of practical importance."

Another probable reason why bronchographies are seldom performed in very young patients is that they are much more difficult than in adults.

To execute a correct filling and succeed in making serviceable roentgenograms requires a perfect technique, very fast work, a tremendous amount of patience and some luck.

The special technical difficulties and the not infrequently poor results are not sufficient reason to neglect or discard a useful diagnostic procedure.

The lack of enthusiasm of most endoscopists when asked to perform a bronchography in a child seems to be shared by most pediatricians. They either do not appreciate the possibilities of bronchographies in many doubtful lung cases in their little patients or feel dubious about its application. These psychological factors added to the unsatisfactory results obtained explains the discouragement of the early investigators.

CONCLUSION

No one will contest the necessity of a thorough examination in difficult or doubtful chest cases. No one will deny the usefulness of a bronchography in those cases or assert that it is profitless in young children. The special difficulties of these techniques or the eventually poorer results when applied to infants and young children as compared with adults, must not deter us from trying and using them whenever indicated.

It should on the contrary incite us to persevere in our efforts to improve our x-ray technique and perfect our team-work.

I am positive that due to the patient efforts of the endoscopists, their persuasive power and the encouraging results they will achieve, bronchography in infants and young children will get the impetus it needs and deserves.

AVENUE DE L'YSER, 12.

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LXXI

AQUEOUS CONTRAST MEDIA IN BRONCHOGRAPHY

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AND

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Thirty years have now elapsed since the initial demonstration of iodized oil instillation as a practical method of bronchography. Interestingly, the preparation first employed, Lipiodol, had been used therapeutically for nearly twenty years, and although instillations in cases of bronchial infection were practiced as early as 1904,[1] the suitability of iodized oil as a contrast medium for bronchography was not recognized until the investigations of Sicard and Forestier in 1921.[2] Meanwhile, bronchoscopic insufflation of bismuth powder had been employed by Chevalier Jackson[3] as a means of demonstrating the larger bronchi.

Of the oily preparations now in use, iodized poppyseed oil (Lipiodol) is probably the most widely employed. Though providing maximum contrast through its high iodine content (40%), Lipiodol is somewhat less stable on exposure to heat and light than Iodochlorol (27% iodine and 7.5% chlorine in organic combination with peanut oil). Either of these substances, if swallowed, may liberate free idodine on digestion in the intestine, so that manifestations of iodism, usually mild and of short duration, may be seen; delayed urticaria is occasionally observed. Severe or even fatal immediate reactions, attributed to anaphylaxis, are reported in rare instances, though in some of these differentiation from the toxic effects of the local anesthetics used would seem to be difficult.

In practical use, prolonged alveolar retention of iodized oil has been disadvantageous in interpretation of serial roentgenograms. Histologic changes, consisting in macrophagic response, both alveolar and interstitial, are thought to be reversible and of little consequence in most cases, although, as will be discussed below, there is no general agreement on this point. The occasional cases of "lipoid pneu-

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monia" and iodized oil granuloma constitute an objectionable feature of greater importance.

The theoretical advantages of a medium which would be more rapidly eliminated and elicit a minimum of tissue response has been recognized for many years. Stable water-soluble organic compounds of iodine have been available, but simple aqueous solutions of these substances were of such low viscosity and so irritating as to be impractical. Attempts to increase viscosity by use of such substances as pectin, acacia, tragacanth, gum arabic, polyethylene glycol and polyvinyl alcohol were not successful because of incompatibilities with the contrast substance, high concentration required to produce the desired viscosity, or excessive irritation.

Morales and Heiwinkel[4] of Stockholm investigated the possibility of using sodium carboxymethyl cellulose as a viscosifying agent; when added to aqueous solutions of Iodopyracet, a contrast medium of sufficient viscosity and iodine content for bronchography was produced. Their observations on the clinical use of this preparation, now manufactured under the trade-name "Umbradil Viscous B," were first published in October, 1948. Soon afterward Fischer,[5] in Zurich, reported results with a similar preparation, Ioduron B. Both Morales[6] and Fischer[7] have published subsequent reports based on further clinical trial.

Umbradil Viscous B* has the following composition:

Umbradil (Brand of Iodopyracet) (diethanolamine salt of diiodo-pyridone acetic acid)		50.0 Gm
Carboxymethyl cellulose		q. s.
Xylocaine (diethyl-amino-aceto xylidide)		0.5 Gm
Sterile Distilled Water	q. s. ad	100. ml

Xylocaine,[8] an acetanilide derivative and thus chemically unrelated to cocaine, is a relatively new local anesthetic agent, said to be more effective than other agents of corresponding toxicity (maximum dose 0.5 Gm).

Ioduron B** is made according to the following formula:

Ioduron (Brand of Iodopyracet) (diethanolamine salt of diiodo-pyridine acetic acid)		50.0 Gm
Carboxymethyl cellulose		2.5 Gm
Wetting Agent (Nicotine acid amide N-cetyl bromide)		
Preservative (p-oxybenzoic acid ethers)		
Sterile Distilled Water	q. s. ad	100. ml

*Product of A. B. Astra, Sodertälje, Sweden.
**Product of Cilag, Ltd., Schaffhouse, Switzerland.

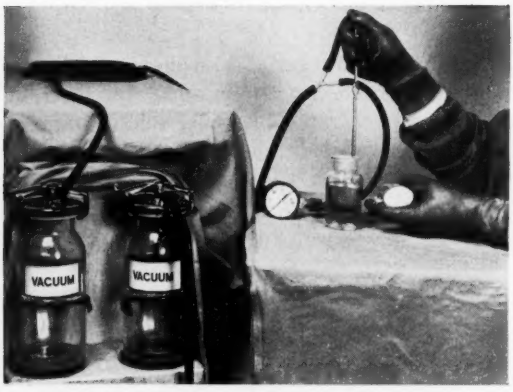

Fig. 1.—Simple apparatus for comparing viscosities of media used in bronchography. Viscosities are in the ratio of times required for filling of the 1 ml pipette, with constant negative pressure of about 300 mm Hg.

The "wetting agent," by decreasing the surface tension of the solution, is employed to facilitate spreading over the mucosal surface.

In their essential constituents, Umbradil Viscous B and Ioduron B are identical. Since Iodopyracet contains 49.8% iodine, the net iodine content of both preparations is 24.9%, slightly less than that of Iodochlorol. Following intrabronchial instillation, this substance is rapidly absorbed (usually in less than four hours, as demonstrated by roentgen study) and is eliminated by renal excretion. The carboxymethyl cellulose component is thought to be largely eliminated by cough and the other normal mechanisms of bronchial drainage, the remainder being removed by phagocytosis. Hellstrom and Holmgren[9] found only traces of the material histologically three weeks after intrabronchial instillation of relatively large doses in rats.

The viscosity of Ioduron B, as supplied, is much greater than that of Umbradil Viscous B. Measurements by means of a vacuum-pipette method (Fig. 1) indicate that the viscosity of Umbradil is about half, and the viscosity of Ioduron about 6 times that of Lipiodol (Fig. 2). Bronchograms obtained following instillation of Umbradil accordingly are more likely to show peripheral and alveolar filling, and will show mucosal coating rather than bronchial filling. Morales[6] feels that this type of bronchogram is preferable for diagnostic purposes. Fischer,[7] however, whose work has been with the medium of greater viscosity, is convinced that more complete filling is desirable, at least for routine work.

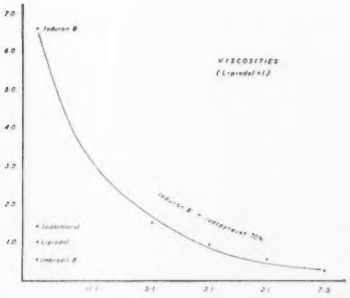

Fig. 2.—Relative viscosities of aqueous and oily media uesd for bronchograpy. For convenience, the viscosity of Lipiodol is used as the standard.

It would appear that no one viscosity will be suitable for all cases. In fact, one of the advantages of the aqueous media lies in the ease with which their viscosity may be regulated over a wide range. This may be accomplished by adding carboxymethyl cellulose to Umbradil, as practiced by Atwell and Pedersen,[10] but the same result may be accomplished more easily by adding an aqueous solution of Iodopyracet (such as Diodrast[r] 70%) to Ioduron B to obtain the desired viscosity. This decreases the concentration of the viscosifying agent and slightly increases the concentration of the contrast substance. Viscosities of various combinations of Ioduron B and Iodopyracet 70%, using Lipiodol as the standard of comparison, are shown in the accompanying graph (Fig. 2). Ioduron preparations of lower viscosity are also made.

For general use, the best aqueous preparation has appeared to be one having a slightly greater viscosity than that of Lipiodol. Movement of this medium into the peripheral portion of the bronchial tree is slower than in the case of Lipiodol, so that more time is available for systematic instillation under fluoroscopic guidance, with a minimum of alveolar filling. Media of greater viscosity are useful in demonstrating deformities of the larger bronchi by thick coating or complete filling (Figs. 5 and 6).

TECHNIQUE

In principle, the technique of instillation is identical with that described by Jackson and Bonnier[11] before the American Broncho-Esophagological Association in 1937. Differences in detail are chiefly those relating to the application of topical anesthesia; the aqueous media, being hypertonic, are much more likely to produce cough

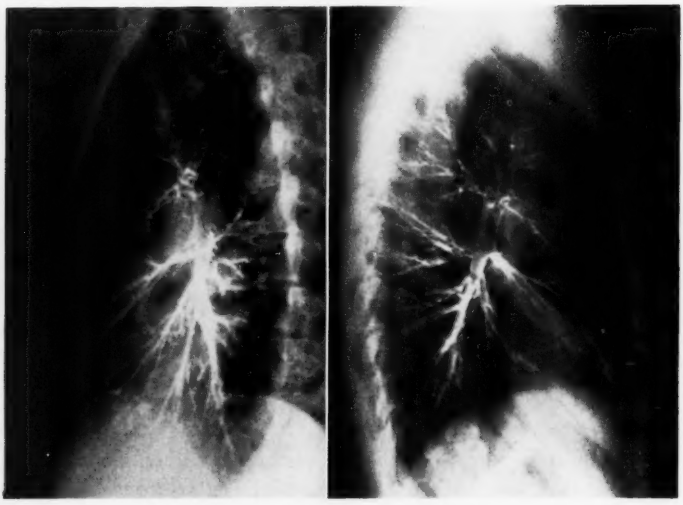

Fig. 3.—Normal right bronchogram (Ioduron B) in (a) left anterior oblique and (b) right lateral projections.

than the iodized oil. This does not necessitate the administration of a large or unsafe dose of anesthetic agent, but does require more uniform and diffuse distribution of the solution employed. This is accompanied by the instillation of small portions of more dilute anesthetic solution through the accurately positioned catheter with the patient in a series of successive postures to allow flow into various parts of the bronchial tree. As the dilute anesthetic solution, a mixture of equal parts of pontocaine hydrochloride ½% and xylocaine hydrochloride ½%, as suggested by Fischer[7] appears to be well tolerated and effective.

As premedication, the average patient is given codeine sulfate 30 mg and atropine sulfate 0.4 mg, both hypodermically, and sodium pentobarbital 100 mg by mouth about 45 minutes before the procedure. Initial topical anesthesia is applied with the patient sitting on the edge of the fluoroscopic table. The pharynx is sprayed lightly three or four times with 4% solution of cocaine hydrochloride, following which 3 ml of 1% pontocaine hydrochloride solution are instilled fractionally with a laryngeal syringe under mirror guidance, favoring gravitation into the bronchial tree of the side to be filled by having the patient incline his body after each instillation. The catheter is then introduced on a stilet under mirror guidance and positioned fluoroscopically so that its end lies at, or just proximal to,

the upper lobe bronchial orifice of the side to be filled. The patient is then turned to lie on the apropriate side, and 1½ ml of pontocaine xylocaine mixture is instilled through the catheter; this instillation is repeated with the patient in the antero-lateral and postero-lateral positions. Additional instillations of 1 ml of the same solution are given with the patient sitting, leaning successively forward, to the side and backward. The total dose of pontocaine, slightly less than 0.05 gm, is equivalent to 2½ ml of a 2% solution, an amount consistent with the quantity commonly used for various endoscopic procedures.

If the entire lung is to be demonstrated, the upper lobe is ordinarily filled first, with fluoroscopic observation, the patient assuming in order the positions above described as the medium is injected. Then, as the fluoroscopic table is elevated, suitable postures to favor filling of the lower portions of the bronchial tree are employed. Serial films made during the course of the filling (Fig. 4) are of value, since occasionally filling of upper portion of the bronchial tree will not be maintained until the procedure is complete. These may be obtained by a "spot" film device attached to the fluoroscopic screen, or by means of an overhead tube mounted above the fluoroscopic table. If a Trendelenberg position is to be used to fill the apical portion of the upper lobe, the upper lobe bronchus should be catheterized, using a curved catheter such as that of Metras; otherwise reflux into the trachea may precipitate cough. Overflow into the contralateral bronchus must be avoided for the same reason. Once coughing occurs, it can rarely be controlled well enough to allow completion of the examination. About 20 ml of the contrast medium are required for the average unilateral filling. Routine films should include both lateral and postero-anterior projections, the latter preferably stereoscopic. If the left side has been filled, the right anterior oblique projection is of particular value in demonstrating the lingula. On completion of the procedure, the patient is encouraged to cough out as much of the instilled material as possible.

DISCUSSION

Of the advantages offered by the aqueous preparations, undoubtedly the most important is rapid absorption of the contrast substance. This property is of particular value in the bronchographic investigation of obscure pulmonary disease, when serial roentgen studies are indicated (Fig. 7). A similar application is in the bronchographic localization of foreign bodies, when subsequent fluoroscopic aid will be required for bronchoscopic removal (Fig. 8). In other instances, where the initial bronchograms are unsatisfactory because of cough, excessive alveolar filling or incomplete filling, the

Fig. 4.—Serial films illustrating filling of left bronchial tree (Ioduron B). Stenosis (or spasm?) at branching of lingular division bronchus, left upper lobe. Cylindrical bronchiectasis left lower lobe. All films in right anterior oblique projection.

procedure may be repeated the following day without interference by residual opaque material. On such occasions, use of a preparation of lower viscosity may facilitate complete mapping in the presence of a partially obstructive bronchial lesion or retained secretions, with which the aqueous preparations are miscible.

Chief among the disadvantages is the more exacting technique required for adequate anesthesia. Transient bronchial irritation, with resultant cough, is greater than that following iodized oil instillation, but is perhaps useful in more completely eliminating the non-absorbable component of the mixture. Roentgen contrast is poorer than that of Lipiodol, but only slightly less than that of Iodochlorol. In adults with limited pulmonary function, the preparations of higher viscosity cannot be used because of their obstructive effect. Usefulness in infancy and childhood is limited by the risks of local anesthesia, except in those clinics where general anesthesia is

employed for bronchography in this age group. Bouvet[12] has used Ioduron B in infants and young children anesthetized with ether or ether-chloroform, though the instillation of a substance as viscous as Ioduron B into the bronchial tree under such circumstances would appear to entail some risk, particularly if facilities for bronchoscopic aspiration are not immediately available.

Clinical use of the two preparations described has been considered to involve little risk of toxic reaction. As concerns systemic effect, this is undoubtedly true. Stability of the contrast substance, Iodopyracet, is such that decomposition to yield free iodine does not occur in the lung, gastrointestinal tract or blood stream. Blood concentrations of Iodopyracet following intrabronchial instillation, with absorption over a period of two to four hours, must be much lower than those occurring in intravenous urography and cardioangiography, where a comparable amount of the substance is given intravenously within a few seconds. Advanced renal disease may contra-indicate use of Iodopyracet by any route. Single doses of carboxymethyl cellulose appear to be without toxic effect when given intravenously (Hueper[13]); elimination when given by this route is by renal excretion. The chemical and physical properties of this substance are comprehensively reviewed by Brown and Houghton.[14] The degree and duration of local tissue response following instillation of the various substances used for bronchography is of great importance and will be reviewed in some detail.

EFFECTS OF CONTRAST MEDIA ON THE LUNG PARENCHYMA

Comparisons of toxic effect of the oily and aqueous preparations are significant as a basis for judging the safety of their use. In the case of the iodized oils, variable findings have been recorded, both as regards experimental study in animals and pathologic examination of human specimens.

Fried and Whitaker[15] examined lung specimens of 44 cats after intratracheal instillation of iodized poppyseed and sesame oils. Almost at once beginning proliferation of the alveolar lining cells(the so-called respiratory epithelial cells) was noted, along with the presence of phagocytosed lipoid substance in the cytoplasm. That these cells have a macrophagic function is generally recognized. Proliferation of alveolar cells to form large cellular masses resulted in localized destruction of lung parenchyma. However, in none of the animals, sacrificed from 30 minutes to four months after the instillation, were gross lesions suggestive of pneumonitis or other inflammatory process observed. Microscopically, the reaction reached a maximum in a few days, and resolution occurred in an average of about one

Fig. 5.—Stenosis of left bronchus (squamous cell carcinoma) and advanced bronchiectasis left lower lobe (Ioduron B). Proximal portion of left main bronchus appears normal.

month, without ensuing fibrotic changes. Similar findings in experimental work with cats were reported by Brown.[16] Chesterman[17] observed an appreciable incidence of "lipoid pneumonia," both focal and lobar, following instillation of Neohydriol in cats.

Ballon and Ballon[18] found no early reaction following Lipiodol instillation in rabbits, and there was no recognizable inflammatory response to the residual iodized oil. Gower and Gilmour[19] likewise found no macrophagic reaction or phagocytosis shortly following instillation in rabbits, although epithelioid cell formation was observed later on. Bettman, Kelly and Crohn[20] examined lungs of 12 dogs sacrificed from a few days to seven months following instillation of 5 to 20 ml of iodized poppyseed oil. They found no early cellular reaction attributable to the oil, and no subsequent foreign body reaction or fibrosis was identified, despite persistence of residual oil up to seven months.

The equivocal nature of the above findings would indicate that although preliminary tissue response to iodized oil instillation (macrophagic response and phagocytosis, with alveolar cell proliferation, thickening of alveolar septa and occasional "lipoid pneumonia") may occur in the healthy lungs of animals, resolution proceeds without any permanent damage to the parenchyma.

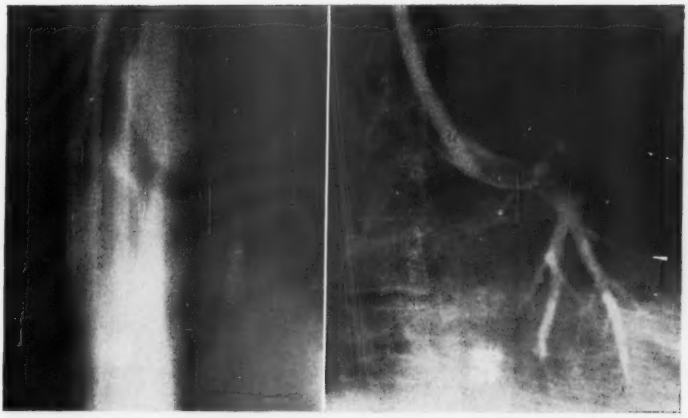

Fig. 6.—Adenoma of left bronchus, 18 years following initial bronchoscopic treatment (C. L. Jackson). Planigram suggested marked narrowing of left main bronchus, (a), but bronchography with Ioduron B demonstrates left main and lower lobe bronchi of normal caliber (b).

The response of the human lung parenchyma to iodized oil instillation is even more difficult to evaluate. Surgical specimens, already diseased, are the chief source of material available to the pathologist, and the reaction of tisue to endogenous lipoid material resulting from degenerative changes in the lung tissue itself is difficult to distinguish from that which might be caused by iodized oil, since both may be of a granulomatous character. In Brody's[21] case of "focal lipid granulomatosis" one month following instillation of iodized poppy-seed oil, the greatest concentration of lipoid material demonstrated histologically was found to correspond to areas of radiopacity in thin slices of tissue, and this correlation was the basis for ascribing the granulomatous changes to iodized oil, rather than edogenous lipoids. Storrs, McDonald and Good[22] employed the same technique in establishing their case of iodized oil granuloma three weeks following instillation of Iodochlorol. In an earlier case, Clagett and McDonald[23] found lipoid pneumonitis in a specimen removed for bronchiectasis following bronchography. Although the changes here were thought to be induced by endogenous lipoid material, the presence or absence of residual radiopaque material in the surgical specimen is not recorded.

Other cases in which granulomatous response has been attributed to iodized oil are those of Wright[24] and Fortner and Miles.[25]

Fig. 7.—(a) White male, 48 years, with pneumonitis of obscure etiology; tumor-like density in right lower lobe. (b) Bronchogram (Ioduron B) nine days later, showing cylindrical bronchiectasis right lower lobe. (c) X-ray four days following bronchography, showing complete absence of residual contrast medium and almost complete resolution of pneumonitis. Diagnosis: cylindrical bronchiectasis with superimposed pneumonitis.

Cases of "lipoid pneumonitis" ascribed to iodized oil have been described by Rabinovitch and Lederer[26] and Fortner and Miles.[25] However, the paucity of such cases in comparison with the tremendous number of cases in which iodized oil bronchography is performed, together with the somewhat equivocal nature of the experimental and clinical data, lead one to conclude that the occurrence of significant reactions and permanent parenchymal damage must be quite rare.

The response of lung parenchyma to the aqueous preparations has been studied in experimental animal work by Hellstrom and

Fig. 8.—Bronchography with aqueous medium (Umbradil Viscous B) in localization of bronchial foreign body. Common pin in medial basal segment, right lower lobe. Removed the following day with biplane fluoroscopic guidance (C. L. Jackson).

Holmgren[9] and by Vischer[27] The former authors, following intratracheal instillations of Umbradil in rats and rabbits, found changes similar to those following Lipiodol instillation, except that these were of much shorter duration and almost completely reversible within three weeks. Staining with toluidin demonstrated phagocytosis of carboxymethyl cellulose, beginning immediately, but only small quantities of this material were identified in macrophages at at the end of the test period. Vischer,[27] in a similar experiment using Ioduron B, found more severe changes of greater duration, with the occurrence of pneumonitis and granulomas.

Evaluation of changes in human lungs, following instillation of the aqueous preparations, is again confused by the apparently frequent occurrence of granulomatous changes due to end products of lung degeneration liberated in chronic suppurative disease. Furthermore, a lipoid or cholesterol type of pneumonitis, unassociated with bronchial stenosis or bronchiectasis, has been described as an entity.[28, 29]

Vischer[27] of Basel, in a recent paper based on pathologic examination of six surgical specimens obtained from 6 to 90 days following Ioduron bronchography found changes of granulomatous type attributed to the presence of residual carboxymethyl cellulose. Zol-

linger[30] of Zurich, in a later publication based on examination of 70 surgical specimens, of which 25 were bronchographic (Ioduron) and 45 non-bronchographic, points out that the stain used by Vischer to identify carboxymethyl cellulose, toluidin blue, does not differentiate this substance from ordinary mucin. The staining technique selected by Zollinger (periodic acid-Schiff reagent) was found to stain mucin but not carboxymethyl cellulose, and Zollinger concludes that the lesions described by Vischer do not represent response to any instilled substance, but are rather "mucin-granulomas," found in both bronchographic and non-bronchographic diseased lungs. It is worthy of note that even Vischer does not feel that the changes induced by Ioduron bronchography are more severe than those following instillation of iodized oil.

The effect of iodized oil bronchography on pulmonary function has been investigated by Zavod,[31] who found some degree of temporary diminution in the pulmonary volumes in 90% of the patients without long-standing or extensive pulmonary disease. Return to normal levels occurred within five days or less. According to Fischer, slightly diminished oxygen saturation is observed for 24 to 36 hours after bronchography with Ioduron B.

SUMMARY AND CONCLUSIONS

1. Observations on the properties and uses of two aqueous contrast media for bronchography (Umbradil Viscous B and Ioduron B) are recorded.

2. On comparison with the iodized oils, the aqueous preparations offer the advantages of rapid elimination of the contrast substance (Iodopyracet), chemical stability, viscosity which may be varied and freedom from systemic toxic effect. Local tissue reaction is probably of lesser degree and shorter duration, although this remains to be conclusively proved.

3. Technically, the aqueous media are more difficult to use, chiefly because of their greater, though transient, irritant effect. This requires a well distributed and more complete topical anesthesia.

4. Details of technique are described and illustrative bronchograms are presented.

5. The literature describing effects of oily and aqueous preparations for bronchography on the lung parenchyma is briefly reviewed.

6. The advantages of the aqueous media should not be overemphasized. Bronchograms made with these preparations have not in general been more informative than those obtained with iodized

oil, and in some cases appear less so. Although the special applications described above may be regarded as indications for their use, general adoption for routine bronchography will depend on ultimate demonstration that tissue response is of significantly smaller degree than that occurring following instillation of iodized oil. Further studies of surgical and post-mortem specimens following bronchography would appear to be the most logical method of investigation.

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DISCUSSION

DR. WHELAN (Stockholm, Sweden): In Sweden, we were not satisfied with lipiodol as a contrast medium. Two of my colleagues were able to produce a new contrast medium, umbradil. I have been able to do 500 examinations with this contrast medium without any complications.

(Slide) I have here to the left contrast medium in the lung and one hour later nothing is seen in the next roentgenogram. In the beginning, we worked with different degrees of viscosity of the contrast medium. We came to the conclusion that the present relatively easier flowing liquid was more suitable. We were able to outline the mucous membrane if we so wished.

If we want massive filling of the bronchial tree, as at times is desirable in bronchiectasis or tumors, we inject more of the contrast medium, as much as 50 cc of the preparation can be injected without causing the patient any discomfort.

If we increase the viscosity, as has been done with certain foreign preparations, massive filling is obtained from the beginning. The question arises, if it is possible to obscure minimal changes of the mucous membrane under such conditions, and if it is absolutely harmless to inject such a slow flowing preparation

into the bronchi. From my experience, I think that it is advantageous in many cases merely to coat the mucous membrane and we have been striving to obtain such procedures.

I would like to show some examples of this.

(Slide) Here we see a case with atelectasis of the upper lobe. The bronchi of the middle and lower lobes are coated with a thin layer of contrast substance, while the middle lobe bronchus is displaced upwards. In the upper lobe bronchus, we have an irregular contour compatible with bronchitis.

The next slide is a roentgenogram of a patient who had sputum positive for tuberculosis. The chest field and the bronchoscopic examination were negative, and therefore the patient was referred for bronchography. On the superior margin of the right upper lobe bronchus is an ulcer of the mucous membrane which explains the clinical findings. With this preparation, umbradil, rapid alveolarization occurs, but does not take place before adequate pictures are obtained.

For example, in this picture the finer bronchi of the upper lobe and middle lobes are outlined, while the lower lobes are not, even though the contrast medium was injected in such position as to favor filling of the lower lobe. This finding is explained by the fact that the examination was carried out during an asthmatic attack with spasm of the lower lobe bronchi.

(Slide) This picture illustrates that alveolarization does not prevent diagnosis being made. This is a case with recurrent attacks of bronchitis. Repeated clinical examinations were negative. Bronchography shows on this spot film those widened areas of the superior branch of the left lower lobe, which is compatible with localized bronchitis.

(Slide) This next patient had chest fields showing only slight bronchovascular markings in the right upper lobe. At bronchography this was found with changes in the bronchus leading to a cavity, and during fluoroscopy I saw the contrast medium run through this bronchus to the cavity.

(Slide) My last case is one of a patient who had sputum positive for tubercle bacilli, but on bronchoscopy revealed no abnormality. The patient was referred for bronchography and as we can see, there is a small cavity in the apex.

As umbradil is a non-toxic preparation which is excreted in a few hours, and inasmuch as we can repeat the examination on the next day if desired, the indications for bronchography have greatly increased. Therefore I am of the opinion that through your excellent bronchoscopic technique, and through this new water-soluble contrast medium, we can look forward to more accurate diagnosis of diseases of the lungs.

JOSEPH P. ATKINS, M.D. (Philadelphia): This very interesting paper brings forth the difficulties which have been encountered with bronchography in the past. I think that we should regard these substances more as a step in the right direction, rather than the final answer. We have tried working with both of them, and have been impressed with the added anesthesia which is necessary to accomplish satisfactory instillation of the material.

There is one comment I would like to make about the matter of bronchography in children. In very small infants, even new born children, bronchography can be carried out without anesthesia primarily because one is interested (in the new-born child) in seeing the larger bronchi; the detail of the smaller bronchi is seldom of importance. In the older child, the detail of the smaller branches of the bronchial tree becomes an important consideration. Dr. Norris is to be congratulated on the excellence of his presentation.

H. M. STAUFFER, M.D. (Philadelphia, Pa.): I would just like to say that the radiologist cannot help but be impressed with a medium that disappears as rapidly as you have seen that this does from the lung. We are always discouraged by the problem of the residue, and differentiating the residual lipiodol from pathological changes in the lung.

There are several things, very briefly, that occur to a radiologist. One of the things that inevitably is going to come up in this country is the question of testing for sensitivity to the iodine compound. There is a pretty general feeling that none of the tests really provide any indication of sensitivity. I feel that it is probably not necessary in this particular field.

Another point that occurred to me, as Dr. Welin was showing his very beautiful films, is the question of the spot films which seem to be often desirable, and very often necessary in this sort of work, because of the rapidity with which the medium moves out into the alveoli. It should be pointed out that the average spot film device on the fluoroscopes that we have is usually designed for gastro-intestinal work at much lower speeds, and you probably won't be getting very satisfactory spot films, unless your devices are arranged for more rapid exposures of the order of 1/20th or 1/30th of a second, usual in chest roentgenography.

LXXII

OBSCURE PULMONARY BLEEDING

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Pulmonary bleeding is a frequent symptom of thoracic disease and usually no mystery hides its source. A survey by Abbott of 1316 patients with hemoptysis, lists more than fifty separate conditions as causes of such bleeding. Of these the three top offenders were: tuberculosis, bronchiectasis, and carcinoma in that order—and comprised, by far, the majority of the 1316. Eliminating tuberculosis from consideration, another survey by Jackson and Diamond[1] of 436 patients with hemorrhage of non-tuberculous origin, the sequence of causes ran as follows:

1.	Bronchiectasis	138	or 31.65%
2.	Carcinoma	82	or 18.81%
3.	Tracheo-bronchitis	74	or 16.97%
4.	Pulmonary Abscess	51	or 11.70%
5.	No Evidence of Disease	34	or 7.80%
6.	Bronchial Adenoma	11	or 2.52%
7.	Miscellaneous	46	or 10.55%

It is very interesting to note that 140 of these 436 patients presented normal-appearing chest x-rays—and also that in 109 of the 436 the site of bleeding was visualized by bronchoscopy.

Until recent years hemoptysis has been considered presumptive, if not conclusive evidence of pulmonary tuberculosis and in the past many patients have been admitted to sanatoria because of this symptom alone. Granted that pulmonary bleeding demands that investigation be directed toward tuberculosis first and foremost, experience shows that other pulmonary pathology must be borne in mind. Andosca and Foley in 1942 found that while hemoptysis between the ages of 20-40 usually meant tuberculosis (with bronchiectasis and mitral stenosis, second and third, respectively), bleeding in patients over 40 was an indication of probable bronchogenic carcinoma.

From the foregoing then, it seems generally agreed that tuberculosis, bronchiectasis, and carcinoma are the most frequently en-

countered causes of pulmonary bleeding. Because tuberculosis and carcinoma present indications other than hemoptysis which help establish a diagnosis, I wish to omit them from consideration in this presentation in order to focus attention on an important group of patients in whom pulmonary bleeding is essentially not only the sole symptom, but sometimes the only sign of thoracic disease.

This group represents the so-called "dry" or "hemorrhagic bronchiectasis," which is encountered more frequently than is generally appreciated. Our medical teaching and literature give scant attention to this condition, which probably accounts for the fact that when we think of bronchiectasis we think in terms of productive cough with profuse, foul expectoration, sometimes blood-streaked, clubbed fingers, and repeated episodes of pneumonitis. Even in such a representative text-book as Cecil,[6] these cases are described briefly in these words: "There is a so-called dry hemoptic type (of bronchiectasis) in which the first symptom is hemoptysis, without any noticeable antecedent cough. In some instances hemoptysis may occur at rare intervals for many years without any other conspicuous symptoms. As a rule such dry types, if untreated, eventually develop chronic cough and expectoration as a result of infection."

Since the introduction of bronchography by Sicard and Forestier in 1922, bronchiectasis has been found to be the second most common chronic pulmonary disease—preceded only by tuberculosis. Largely because of this technical advance in our investigative measures, by which we are able to map out the tracheo-bronchial tree with opaque oil, our knowledge of the disease rather quickly increased. That this knowledge is recent is indicated by the fact that the first comprehensive monograph by Lisa and Rosenblatt[2] on bronchiectasis appeared as recently as 1943.

Statistics and experience emphasize the importance of the diagnosis and treatment of bronchiectasis. It is estimated that there are 2,000,000 cases in the United States. Hawkins and Clagett[3] of the Mayo Clinic report incidence of 2% of bronchiectasis in all autopsies; Bradshaw, Putney and Clerf[4] report 59 deaths (34.5%) in 171 patients with untreated disease and found the average duration of life from onset of symptoms was 13½ years; and Roles and Todd[5] estimate a 47% mortality in 49 non-surgically treated patients followed over six years. Obviously then, measures aiding early diagnosis are desirable for the physician and beneficial to the patient. A review of the literature gave no clear evidence as to the percentage of patients whose predominant, essentially solitary symptom, was hemoptysis. In general, articles simply mention bleeding as an accompanying feature in "more than 50%."

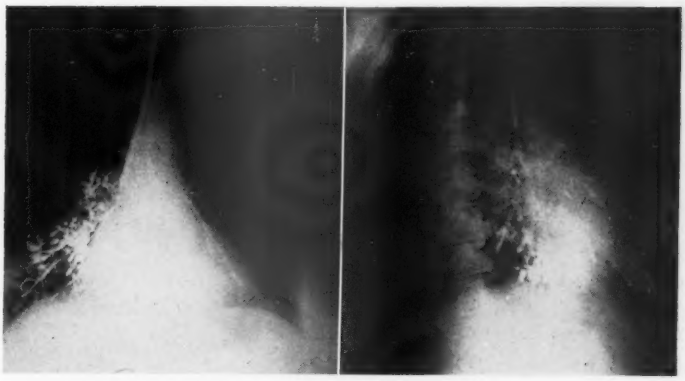

Fig. 1.—(left) Case 1. Roentgenogram showing bronchiectasis of right upper and middle lobes. (right) Case 2. Roentgenogram showing bronchiactasis of left lower lobe.

In summary then, experience would seem to indicate that patients with hemoptysis, and without other symptoms of consequence, and without significant history, physical, and radiographic findings —may be bronchiectatics of the so-called "dry" variety. If tuberculosis and carcinoma are unlikely, and if other lesser possibilities such as mitral stenosis, blood dyscrasias, and vitamin deficiencies are ruled out, investigation by bronchoscopy and bronchography is often rewarding and sometimes conclusive. To be sure there are other entities to be remembered and, while less likely, must not be overlooked. However, experience in the evaluation of cases of obscure pulmonary bleeding has led us to believe that the majority of these patients have unsuspected underlying bronchiectasis—the ectasis preceding the infection phase yet to come—and the diagnosis of which can often lead to happy therapeutic result.

Our foremost aid in the diagnosis of this condition is the knowledge of its existence and the awareness of it as a possible explanation of unexplained pulmonary bleeding. It is unwise to be content with the radiological diagnosis of "essentially normal chest" in the face of hemoptysis. Roentgenograms, per se, are not the ultimate in evaluating pulmonary pathology and this is said with the utmost respect for, and acknowledgement to, this most important branch of our diagnostic armanentarium.

With the realization that this little known type of bronchiectasis exists, and at one time or another confronts all of us, by pool-

Fig. 2.—(left) Case 3. Roentgenogram showing bronchiectasis of right middle and lower lobes. (right) Case 4. Roentgenogram showing bronchiectasis of left lower lobe.

ing our clinical experience, our diagnostic abilities, and our investigative procedures, the condition can be discovered and adequately treated.

For our purposes, the cases presented had episodes of bleeding varying from hemoptysis of a few teaspoonsful to hemorrhages of such extent that replacement therapy was necessary. In all patients, the only symptom of significance was bleeding; the most conclusive means of diagnosis was by bronchoscopy and bronchography, and the most definitive method of therapy was surgical resection.

Case 1.—B. S., aged 49. This patient who has hypertension had no pulmonary symptoms until a mild "virus infection" in Spring of 1950. Beginning in February, 1951, she had small hemoptyses almost daily—usually in the morning. She was admitted to the hospital in March, 1951, where bronchoscopy and bronchography revealed bronchiectasis of the right middle and upper lobes. Because bleeding was not extensive, and, because of obesity and hypertension, it was felt best to avoid surgery at this time. However, after returning home hemoptysis was so persistent that patient was re-admitted and temporary phrenic crush was done with good result.

Case 2.—R. H., age 34. This patient has had episodes of hemoptysis since early youth. X-rayed many times, but films were always negative. The bleeding seemed to occur about one week prior to menses. Her last hemoptysis was in April, 1951. Following this she was persuaded to enter the hospital for investigation. Bronchoscopy showed minor inflammatory changes in the left lower lobe bronchus, and bronchograms revealed bronchiectasis in this area. Patient is now awaiting resection.

Case 3.—I. K., age 70. This patient had so-called bronchitis of many years duration, but never had been disabled or even seriously inconvenienced by the condition. At age 67 for the first time she raised blood estimated by the patient

as "at least a pint." She was hospitalized, transfused, and investigated. Bronchoscopy revealed bloody secretions in the right-stem bronchus. Bronchograms showed cylindrical bronchiectasis of the right middle and lower lobes. Again in March, 1951, patient had a massive pulmonary hemorrhage requiring re-admission and blood replacement. Hypertension was not a factor. No resection contemplated, but phrenic crush advised if bleeding recurs.

CASE 4.—M. C., aged 37. This patient without antecedent symptoms, experienced her first hemoptysis at age 34. Bleeding recurred several times always followed by chest films which were negative. At 39, after an unusually large hemorrhage, she was hospitalized and G.I. tract investigated with no success. No bronchograms at that time. At age 40, following another episode, she was bronchoscoped and lung fields mapped. Surgery was advised for the extensive left lower lobe bronchiectasis, but patient refused. Five years elapsed during which patient had no serious hemorrhage, but during the summer of 1949, rather profuse bleeding led her to decide to accept surgery. The left lower lobe was uneventfully resected in January, 1950, with no further symptoms to date.

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LXXIII

PRIMARY TUBERCULOMA OF THE BRONCHUS

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It is not unusual, in the presence of active pulmonary tuberculosis to observe bronchoscopically the formation and development of granulations of the bronchial mucosa, which, on histologic section, present a picture typical of tissue tuberculosis. Such lesions are properly classified as granuloma, tuberculous, secondary to pulmonary tuberculosis. Nor is it unusual, in the presence of such granulomata to observe the development of pulmonary obstructive phenomena due to emphysema or atelectasis. In fact, the presence of such signs implies the existence of obstructive lesions of this nature.

Conversely, bronchial obstruction, in the absence of discernible roentgenographic signs of pulmonary tuberculosis, is never assumed to be tuberculous in origin. Primary considerations in such instances would be given to benign or malignant bronchial tumors, foreign body lodgement, inspissated bronchial secretions, and to lesions possibly effecting compression stenoses. Mindful of these mechanics of bronchopulmonary physiology, one is properly chagrined on encountering a case that proves contrary to the rule. The following case report serves as a typical example.

R. A., a colored male child, aged seven years, was admitted to the hospital on February 28, 1950. The history, obtained from the child's mother, was one of recurring episodes of pneumonitis, wheezing respirations since early childhood, and inability to withstand the physical efforts ordinarily expended by a child. Repeated Roentgen ray studies of the chest had been made during these periods of respiratory infection, but, other than the diagnosis of pneumonitis, there had been no reference made to the phenomenon of bronchial obstruction. Review of the films discloses the following: Figure 1*a*, made on August 8, 1949, presents a typical picture of obstructive emphysema of the left lung. Figure 1*b*, made on October 12, 1949, presents a picture of diffuse pneumonitis involving the base of the left lung, together with signs of atelectasis of the left base as indicated by displacement of the heart and mediastinum towards the

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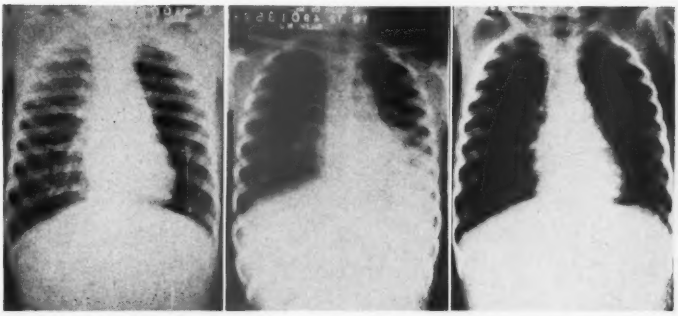

Fig. 1.—Roentgenograms of the chest illustrating successively obstructive emphysema of the left lung; obstructive atelectasis, together with patchy pneumonitis of the left base, and again, obstructive emphysema of the left lung.

left, and elevation of the left diaphragm. Figure 1*c*, made on January 4, 1950, again indicates the presence of moderate diffuse emphysema of the left lung. In none of these films is there any evidence of pulmonary tuberculosis.

Physical examination, limited to the pertinent findings, revealed the following: there was inequality of respiratory movements; the left hemithorax, while being full, was definitely limited in its respiratory excursions; there was an obvious respiratory wheeze, which was present both during inspiration and expiration, but more pronounced during the expiratory phase. On auscultation, this wheeze was defined as originating in the left bronchus and was grossly more pronounced during expiration.

Bronchoscopy, performed on March 1, 1950, is recorded as follows: "The trachea appears to be in the mid line. Inmmediately upon entering the left main bronchus there was seen a large rounded globular mass, causing almost complete bronchial obstruction. It was deep red in color, and covered by an intact, coarsely pebbled mucosa. Its point of origin was on the left lateral wall of the left main bronchus and was apparently pedunculated, in that it moved back and forth with respiration. The mass was easily and completely removed, using forward grasping forceps. Following removal, it was noted that the orifices of the various bronchi on this side were normally patent and functioning. There was little bleeding following removal."

Laboratory studies on the bronchial secretions collected are reported as follows: March 2, 1950—smears negative for acid-fast

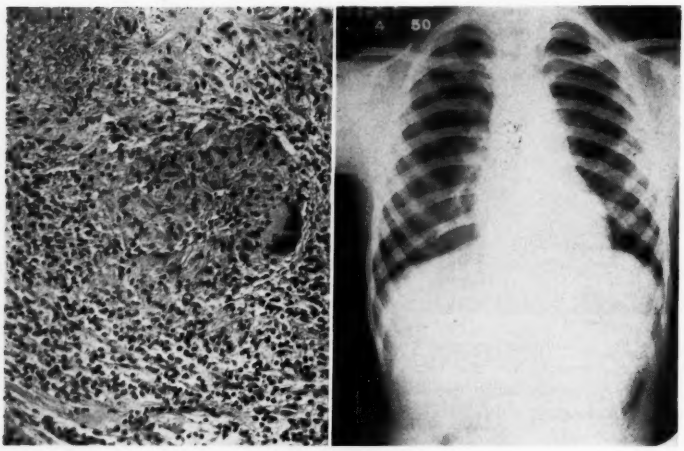

Fig. 2.—(L) Photomicrograph showing the presence of tubercles contained within bronchial granuloma. x200.

Fig. 3.—(R) Roentgenogram of the chest made on April 5, 1950, following removal of the bronchial tuberculoma. The chest is essentially negative; shows no further evidence of bronchial obstruction.

bacilli. Histological study of tissues secured from the bronchus reveals the presence of a chronic granulomatous mass consistent with tuberculous bronchitis." On March 11, 1950, the bacterial culture was reported as "negative for growth of tubercle bacilli."

The child was discharged the day following bronchoscopy and readmitted to the hospital after six weeks' time. Bronchoscopy this date, April 4, 1950, revealed a perfectly normal appearing left bronchus with no evidence of recurrence of the previously noted inflammatory mass. Bronchial secretions secured at this time were negative by smear for acid-fast bacilli and negative by culture for growth of tubercle bacilli. A film of the chest on this admission showed no evidence of bronchial obstruction.

COMMENT

The microscopic study of the tumorous mass unquestionably identifies it as tuberculous in origin; in addition to its typical cellular pattern there are central caseation and demonstrable acid-fast bacilli. On the other hand, the clinical picture, the cultural studies of bronchial secretions, and the Roentgen examinations of the chest, have repeatedly failed to demonstrate the existence of pulmonary tuberculosis.

These facts offer an interesting conjecture. In appreciation of the usual course of events in tuberculosis, however, it is only by way of extended mental reservations that one could classify this granuloma as a primary tuberculoma. For, although the clinical evidence of pulmonary tuberculosis is negative, or lacking, its existence cannot be disproved. Therefore, it must be assumed, for the time being, that this tuberculous granuloma is secondary to an undisclosed primary cause.

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LXXIV

THE APPLICATION OF STREPTOMYCIN IN TUBERCULOUS BRONCHITIS

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The serious potentialities consequent upon the development of advanced endobronchial tuberculosis create in that condition a situation which may justifiably be viewed with alarm unless the progress of the disease can be arrested.

The controlling factors that determine wherein a patient's bronchial disease progresses from the stage of an early inflammation of the mucosa to the development of a strangulating stenosis still remains uncertain or undetermined. It is a fact, however, that many times the sequence of events leading finally to a stenosis of the bronchus may be interrupted at any stage, either spontaneously or through the intervention of some extrinsic factor such as the control of the parenchymal lesion within the lung itself; the endoscopic management of the condition; or the influence of streptomycin as a direct bacteriostatic agent.

The control of the parenchymal lesion continues to be the ultimate if the endobronchial lesion is to remain permanently inactive. Although a certain few cases will develop an ulcerative endobronchitis, or stenosis, in the absence of a demonstrable lesion in the lung tissue itself, the greater majority of inflammatory and ulcerative lesions show a rapidly favorable change upon elimination of tubercle bacilli from the sputum (Case 1, Fig. 1).

Bronchoscopic treatment, with the aspiration of secretions, and the application of either an astringent solution (Silver Nitrate 5%) or a soothing reagent (Monochlorphenol, Guminol) or both, contributes measurably although the exact rationale may not be entirely clear. Those bronchoscopists whose privilege it has been to bronchoscope a considerable number of patients and to follow them on the wards, have noted, undoubtedly, numerous instances of dramatic improvement in patients even after a single bronchoscopy and without an apparent adequate explanation to account for these changes.

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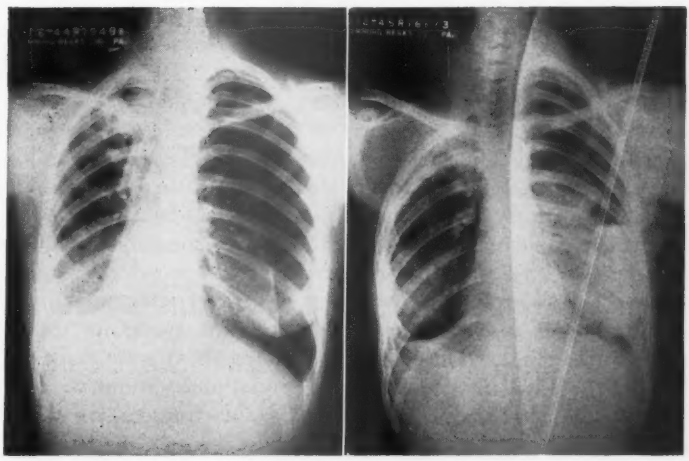


A *B*

Fig. 1, Case 1.—*A.* Preoperative roentgenogram—shows active tuberculous lesion in right upper lobe and pneumothorax collapse of left lung. At this line there was an extensive ulcerating endotracheobronchitis involving trachea, right and left bronchi.

B. Roentgenogram following successful extrapleural pneumothorax and convertion of this to a thoracoplasty on the left side. The effusion on the left side proved to be transatory and easily managed.

The beneficial effect of streptomycin upon endobronchial tuberculosis has been described by several writers. Of those McRae declares that streptomycin is a most efficient method of treatment of tuberculous tracheobronchitis. He further states that in his patients treated with streptomycin and silver nitrate together good results were obtained and similarly as regards those treated with silver nitrate alone. However, he believes that streptomycin showed good results in a much shorter period of time. These observations coincide approximately with the findings observed in this clinic with the exception that we have observed that streptomycin therapy apparently tends to precipitate a stenosis in instances of advanced tracheobronchial disease sooner than in patients treated with silver nitrate alone and under similar circumstances. The suddenness by which a stenosis is precipitated may sometimes preclude effective surgical care and thereby will be instrumental in bringing about a fatal termination of the case (Case 2).

Because of the fact that three of our cases followed such a course and, that two ended fatally, a study of the problem was undertaken

in an effort to discover, if possible, what type of case would yield best to streptomycin therapy and what type of case should be approached with caution.

In the previous communications[1, 2] the opinion was expressed that tuberculous tracheobronchitis should be considered a progressive process and that ulcerations or stenoses themselves do not constitute the ultimate concept of this disease, but rather it would seem better to adapt a dynamic concept of the process as a whole. As a consequence of this belief a classification of tracheobronchial lesions was presented, which it was hoped, could be integrated with existing therapeutic procedures. Following the advent of streptomycin, and the encouraging reports appearing in the literature, its use was adapted in the clinic. The observations noted above were made early following the use of streptomycin and special observations were then undertaken to determine if the application of streptomycin therapy could also be integrated with this classification. A series of cases was studied and comparisons made with a comparable number of control cases. Such a study naturally carries with it certain inherent inaccuracies that cannot be overcome. These inaccuracies are principally those attendant upon differences between individuals in their response to treatment and to disease. As already stated those of us who have enjoyed the privilege of bronchoscoping a reasonably large series of tuberculous patients have, not infrequently, noted outstanding and dramatic changes in cases of endobronchial tuberculosis even following a single bronchoscopy with or without the application of selected medication. It is impossible to know to what to attribute such changes, yet they do occur, and the tendency to attribute these to streptomycin or other agent is difficult to overcome and impossible to isolate in a series being studied such as the one herein presented.

The treatment program consisted of the administration of streptomycin, and later dihydro-streptomycin, intramuscularly, in doses of one gram, three times daily, for 120 days during the early portion of the study but this was later reduced to one-half gram twice daily for a period of not over 60 days. A few cases experienced unpleasant side reactions and drug therapy had to be discontinued as early as 41 days.

When silver nitrate was employed the percentages varied according to the severity of the process, for the non-ulcerating lesions, and those not requiring the removal or cauterizating of granulations 5% and 10% solutions were employed. However, if the cauterizing effect was desired 20% and occasionally but rarely 30% was used.

TABLE I.

GROUP I	Direct Treatment	Parenchymal Treatment	Prognosis For "Healing"
Extrabronchial	None needed in average case	No form of collapse therapy contraindicated Free use of streptomycin	Rapid and satisfactory "healing"
GROUP II Endobronchial	**Direct Treatment**	**Parenchymal Treatment**	**Prognosis For "Healing"**
Type 1 Superficial inflammatory reaction to superficial ulceration not extending into submucosal structure.	Bronchoscopic aspiration, cleaning surface, topical application of Silver Nitrate 5% (non ulcerative cases) Silver Nitrate 10% (ulcerative cases)	Any form of collapse therapy including pneumothorax. Free use of streptomycin	Rapid improvement, complete "healing".
Type 2 Ulcerating lesions extending into submucosal structures; not infrequent weakening of bronchial walls	More energetic treatment; removal of secretions; granulations removed with gauze applicator following by 15% to 30% silver nitrate.	Artificial pneumothorax Thoracoplasty better postponed in Grade 3 cases. Free use of streptomycin except grade 3 cases when caution is advised.	Good prognosis with energetic and somewhat prolonged treatment.
Type 3 Deeper layers invaded, with fixation to actual stenosis or occlusion of lumen	Aspiration Granulations removed by 25 to 30% silver nitrate or actual cautery.	Pneumothorax only with utmost caution. Thoracoplasty (other regional collapse Grade 1 cases. Grade 2 - 3 dangerous. Streptomycin, in grave, with caution. Grade 2,3, utmost caution advised.	Guarded in Grade 1; poor in stenotic cases
Type 4 Healed ulcerating process but stenosis remains	Dilatation of stricture to supply adequate airway and permit proper drainage of secretion	Pneumonectomy or lobectomy in grade 2 and 3 cases or whenever airway is inadequate.	Stenosis remains; dilatations continued indefinitely Considered ultimately unsatisfactory.
Type 5 Miliary tubercle implantations on bronchus or trachea	Bronchoscopic observation: topical treatment dependent upon condition of tubercle	Insufficient number of cases to determine best form of treatment. Free use of streptomycin suggested.	Insufficient number of cases to judge prognosis.

No specific routine was followed regarding the frequency of bronchoscopic examinations. Many patients were subjected to weekly examinations while in others the examinations were performed several weeks apart. The severity of the lesion and the calculated benefit to the patient of such observations and treatments determined the frequency of such procedures.

The cases selected for this study were derived from a series of 558 consecutive tuberculous patients (1134 bronchoscopies). Following the classification previously reported and reproduced here (Table I) in revised form, only those cases conforming with the Type 2 and Type 3 categories need to be included in this report for obvious reasons.

Mention should be made of the action of silver nitrate (5-20%) and of its evaluation as compared with streptomycin in the group

designated "no specific value." Such a comparative evaluation would appear to be somewhat unsound as the one drug, silver nitrate, is primarily employed as a cauterizing or astringent reagent, while the other, streptomycin, is administered specifically for its antibiotic action. Hence, the silver nitrate may be of value in removing granulations and yet not be of value as an assistance in arresting the endobronchial disease. Therefore, it seems advisable to question the implications of that portion of Table II.

The outstanding observation to be noted in Table II centers around:

A. The 7 cases—types 2 and 3 (grade 3 and 1-2 respectively) which suddenly developed a high grade stenosis after receiving antibiotic therapy.

B. Those patients who presented a recurrence of the endobronchial ulcerative change after the discontinuation of the streptomycin therapy.

C. Those who developed refractory ulcerations of the bronchial stump following pneumonectomy or lobectomy.

D. The fact that ulcerative lesions also recurred in the so-called non-streptomycin patients.

E. The longer duration of time noted before the development of a stenosis in those cases not receiving streptomycin therapy.

A. This group illustrates the rapidity of the healing process under the influence of streptomycin and in the presence of conditions which may be considered as representing the optimum. The reason for the sudden stenosis may well be attributed to the rapid formation of scar tissue and subsequent characteristic contraction. It is impossible from our own observation in a small series of cases to predict exactly which cases will and which will not develop such a sudden stenosing lesion. However, the opinion may be ventured that such changes may be anticipated principally in those patients in whom the extent of the tuberculous process extends well beyond the area of ulceration with a widespread involvement of the bronchial lymphatics. This appears to the bronchoscopist as increased rigidity of the bronchial walls with or without actual ulceration. Hence it is suggested that streptomycin be administered with caution in those cases of ulcerative endobronchitis wherein the rigidity of the bronchial walls is marked, irrespective of the degree of visible ulceration.

B. This group presents little difficulty by way of explanation. Although there is an occasional recurrence of the ulcerative process

TABLE II.

GROUP II TYPE 2 GRADE 1-3	TOTAL CASES	RESOLVED WITHOUT PROGRESSING	ADVANCED BEFORE PROCESS BECAME ARRESTED	STENOSIS DEVELOPING 2-3 MO.	STENOSIS DEVELOPING 3-4 MO.	STENOSIS DEVELOPING 4 MO.	PROCESS RECURRING	PROCESS RECURRING AFTER PULM. RESECTION	I.	NO SPECIFIC VALUE NOTED
Streptomycin Therapy	16	10	4	1	0	0	2	1		2
Ag NO_3 Therapy	12	7	5	1	0	0	1			1
No Specific Treatment (All Grade 1 cases)	11	6	5***	0	0	0	2			
TOTALS	41	27	12	1	0	0	5	1		3
TYPE 3 GRADE 1-3										
Streptomycin Therapy	17	13	4	5	1	1**	8	3	3	4
AG NO_3 Therapy	12	7	5	1	3	2	6	4		?*
No Specific Treatment	9	5	4	0	0	1	0	0		0
TOTALS	38	25	13	6	4	4	14	7	3	4

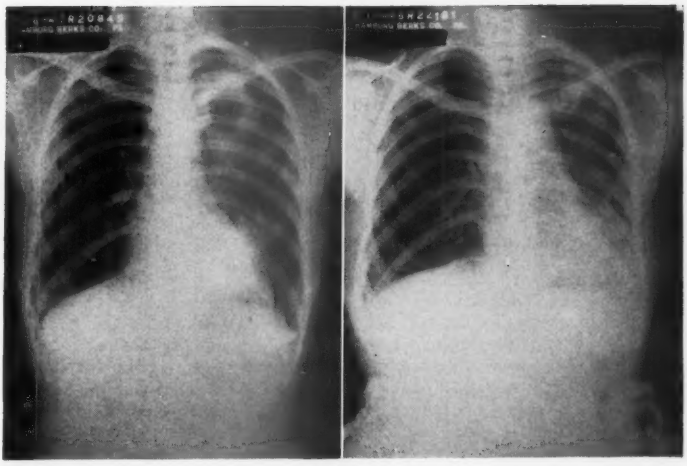
*Explain: We feel that Silver Nitrate being a cauterizing agent and not a specific antibiotic can be evaluated in terms of "no value"
**Stenosis occurred after recurrence of the ulcerative lesion.
***Degree of progression not particularly significant.

in those patients in whom parenchymal lesions have been controlled, and the sputum rendered tubercle bacilli free, the number is small. In reviewing a total of 1058 cases, eleven bonafide instances were encountered. Three developed a gradual stenosis and total resection became necessary. The bronchial stump in each case healed rapidly post-operatively and all three patients are now well and working.

C. This group has created a much more difficult problem to explain. All but one of the resection cases who have developed a recurring post-operative ulceration did not develop this ulceration immediately, but only after a lapse of several months, the longest period being 9 months after the resection (pneumonectomy). At this present moment all have healed without the development of a significant bronchopleural fistula and consequent empyema. The period required for the rehealing of these bronchial stumps has extended from two and one-half to five months. However, all have healed with the additional administration of streptomycin and with silver nitrate applications administered at 3-4 week intervals bronchoscopically.

One additional patient upon whom a lobectomy was performed for a lower lobe cavity developed an ulceration of the bronchial stump nine and a half months post-operatively. There had been no previous evidence of bronchial disease, and the contralateral lung and remaining lobe on the ipsolateral side failed to show previous or subsequent active parenchymal tuberculosis. Why this occurred has not been explained. The ulceration itself extended proximal from the point of the bronchial closure for approximately 1-1.5 cm and "healed" slowly without streptomycin therapy. During the period of the active granulating ulcerations, intermittent positive sputums were obtained on concentrate examinations. The sputum, however, is now negative, (repeated sputum and gastric cultures) and the ulcer is well epithelialized.

D. The fact that bronchial ulcerations recurred in patients not previously treated with streptomycin does not pose a problem for explanation. In our series streptomycin was employed in the majority of the ulcerating cases in the last 558 patients while in a previous group of 500 cases, streptomycin was not available and it was not deemed wise according to the standards of that time to perform a pulmonary resection when the line of amputation passed through active ulcerating tissue. Hence a number of patients were lost who, according to present day practice, would have been brought to operation.



A *B*

Fig. 2, Case 2.—*A.* Admission x-ray film showing large upper lobe cavity with a partial collapse of lung by pneumothorax.

B. Same after lung had fully reexpanded. Note "Tennis Ball" characteristics of the upper lobe cavity. Bronchoscopy revealed an active ulcerating tuberculous bronchitis. Streptomycin therapy instituted.

E. The difference in the time between the development of a stenosis in the streptomycin treated and the so-called untreated cases is, in all possibility, attributable to the fact that the streptomycin hastened the development of the scar tissue through its suppressing action upon the active infective component while in the so-called "untreated" cases, the intrinsic body resistance only provides the principle factor of resistance. It is acknowledged that the sequence of events leading to the final conclusion in such cases is generally slow. This conclusion being either a "healing" with or without stenosis, or a continually progressive ulceration with advancing stenosis of the bronchus or trachea. A like stenosing effect is noted in the treatment of endobroncheal gummas when rapidly healing drugs as potassium iodide and the arsenicals are employed therapeutically.

It is of interest that no significant difference was observed between those patients who received streptomycin three grams daily for shorter periods of time, i.e., 60 or 41 days respectively. The group not receiving specific therapy are recorded for purposes of accuracy although any great measure of significance cannot be at-

tached to these cases. This group includes mostly those individuals where specific therapy was not deemed especially necessary. Hence, the results should, and do, appear superior to those in other categories.

CASE 1.—J. M., age 17, admitted June 15, 1943, discharged October 19, 1945.

This patient was admitted for major surgical work after a satisfactory period of sanatorium care at another institution extending from June 20, 1940 to June 15, 1943. She stated that she had been in good health until January, 1940, after which she became subject to fatique, developed cough, and expectoration, pain in the right thorax, (with subsequent effusion) but no significant weight loss, hemoptysis, night sweats, chills or fever.

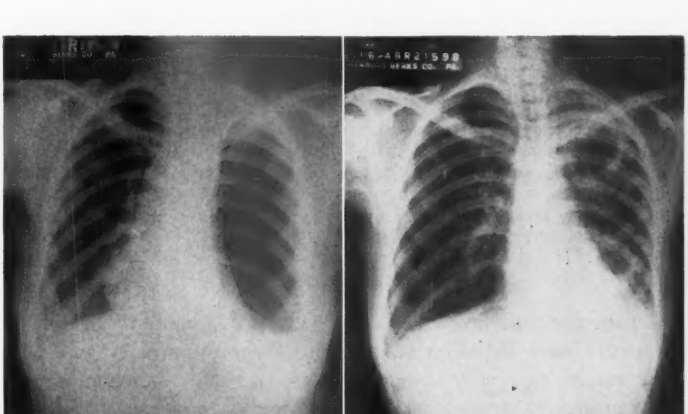
Attempts to establish a right artificial pneumothorax had failed. However, left artificial pneumothorax had been established satisfactorily and maintained by weekly refills until admission here. During the routine preoperative studies the bronchoscopic examination revealed an extensive ulceration involving the right main stem bronchus, 1.5 cm of the left stem bronchus and the lower one-third of the trachea longitudinally and one-half of the circumference, the anterior area being free from visible involvement.

Laboratory examinations throughout revealed no significant findings except for the presence of large numbers of acid-fast bacilli in the sputum.

Streptomycin was not available at this time and the only available direct treatment of active ulcerating tuberculous bronchitis was through repeated applications of silver nitrate applied topically through the bronchoscope, which was without avail. The process showed steady but gradual progression even after 30 endoscopic treatments. Although previous experience with collapse therapy (pneumothorax and surgical) in the presence of endobronchial conditions of this type and degree had been quite disappointing, it was decided that an attempt should be made to control the active parenchymal lesion (right upper lobe) as the patients physiological status, though gradually declining, still rendered her a fair surgical risk.

An extrapleural pneumothorax was established under cyclopropane anaesthesia September 16, 1944. A bronchoscopic examination performed October 31, 1944 showed complete epithelization of the ulcerating area, and at the final bronchoscopic examination, completely satisfactory healing was reported on. No recurrence of the ulceration has occurred. The patient was last seen on July 21, 1950. Since the original operation the extrapleural pneumothorax has been converted to a thoracoplasty, complete recovery followed and the patient is now fully active, employed, and apparently cured in all respects.

Although the rapidity of the epithelization of the ulcerating area borders upon the dramatic the resolution of the process illustrates clearly the value of controlling the parenchymal infection. This patient was subjected to 30 bronchoscopic treatments over a 15 month period with gradual but steady progression of the endotracheobronchitis, yet within six weeks after the elimination of tubercle bacilli from the sputum, and the trauma inflicted by a chronic and troublesome cough, complete epithelization of the process occurred and satisfactory resolution of the entire process without stenosis, was realized within a maximum of three months postoperatively.



C *D*

Fig. 3, Case 2.—*C.* Same after 60 days' streptomycin—cavity closed (stenosis of smaller bronchus, suggested) Bronchoscopic examinations revealed high grade stenosis of left main stem bronchus. Patient considered unfit for any surgical operation because of cardiac status.

D. Same—in terminal stages. Right lung still clear. Cardiac status very bad, spontaneous pneumothorax, bronchopleural fistula, and mixed tuberculous empyema present.

Typical of the seven cases in which a high grade stenosis developed, following streptomycin therapy, is the following:

Case 2.—F. G., age 36, female, was admitted September 24, 1947, with far advanced bilateral pulmonary tuberculosis, and an ineffectual left artificial pneumothorax. The left pneumothorax was ordered discontinued by the staff in conference, and the lung re-expanded preparatory to an intended thoracoplasty. However, pre-operative examination revealed cardiac changes that contraindicated thoracoplasty or any other major surgical procedure at this time. Bed rest and the sanatorium regimen were continued until the patient's condition showed improvement sufficient to warrant further consideration of a surgical procedure. Prior to the cardiovascular study, a routine pre-operative bronchoscopy was performed. This revealed a trachea markedly inflamed in its lower third and inflamed right and left main stem bronchi classified Type 2, Grade 2.

May 23, 1948—The bronchial condition had become worse (Type 2, Grade 3).

April 6, 1948—Streptomycin therapy had been started. The process had advanced further, and narrowing of the bronchus was noted (Type 3, Grade 1).

April 14, 1948—Marked improvement in the inflammatory condition was noted, however, the bronchus remained narrowed (to Grade 1).

April 26, 1948—The bronchoscopic examination showed a very marked improvement in the condition of the mucosa with disappearance of granulations,

epithelization of the ulcerating area, but with a very marked reduction in the diameter of the bronchial lumen.

May 19, 1948—The improvement in the mucosa continued but the stenosis approached strangling proportions.

Pneumonectomy was now indicated, but the patient's clinical condition contraindicated such an operation. In brief, thereafter, the patient developed in turn, spontaneous pneumothorax; a left tuberculous empyema; a mixed tuberculous empyema; contralateral cavitation; recurrence of bronchial ulceration; laryngeal ulcerations; progressive dyspnea; a progressive downhill course and death (Fig. 2 and 3).

COMMENT

From the endobronchial standpoint six other patients followed this same course although the details of the pulmonary and clinical course varied, especially in one, a colored patient, in whom the onset of the bronchial stenosis was so sudden and complete that the consequent massive infection in the obstructed lung catapulted the patient into such a state of toxicity and physiological collapse that all consideration of pulmonary resection had to be abandoned and the patient died.

DISCUSSION

This study was undertaken to determine the value of streptomycin in the management of tuberculous endobronchial disease and to determine, if possible, what the limitations of such treatment might be. Likewise, to determine if this type of therapy could be integrated with the classification previously described.

In consequence of the observations made upon this series, it appears justified to conclude, that streptomycin (or dehydrostreptomycin) constitutes a valuable adjunct to our present forms of treatment.

Streptomycin may be administered with apparent impunity in Group II, Type 1, Grade 1-2-and 3, and in Type 2, Grade 1-2. However, cases falling into the Type 2, Grade 3, and Type 3 (all grades) should be followed very closely lest a sudden stenosis develop and a bronchial strangulation result. Such a strangulating lesion might well prove fatal should it escape attention for any undue length of time or should it occur in a patient in whom a major surgical program must be postponed. There is little if any doubt, but that healing and epithelization result more rapidly and in a somewhat higher percentage of cases than with other forms of treatment.

A number of stenotic lesions were encountered not only in Type 3, Grade 3, but also Type 4, Grade 2-3. In these instances attempts to maintain an adequate airway by bouginage again proved

of no avail over a prolonged period of time. This observation, a by-product of the study, again substantiates the observations of others as well as those made in this clinic throughout the past ten years.

In conclusion, we feel that although streptomycin is an extremely valuable adjunct in the treatment of tuberculous endobronchitis, it is not a 100 per cent curative. It has its limits even when curative, but is curative in a greater number of cases than any other method now available except the satisfactory control of the parenchymal lesion.

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LXXV

CYTOLOGIC STUDIES AND PROGNOSTIC RESULTS IN BRONCHOGENIC CARCINOMA

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AND
T. F. NEALON, JR., M.D. (By Invitation)
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A preliminary report was made before this Association in 1946 on the diagnosis of bronchogenic carcinoma by examination of bronchial secretions.[1] Our results showed conclusively that a tissue diagnosis of carcinoma of the bronchus could be made in cases which were presumably negative on the basis of a bronchoscopic examination. The opinion was expressed that this method would permit of earlier diagnoses of lesions that otherwise would be overlooked and the prognosis of bronchogenic carcinoma should become more hopeful.

This method has been in continuous use in the Department of Broncho-Esophagology at the Jefferson Hospital since that report was made and considerable data have been accumulated. Sufficient information also has been secured by the Surgical Division under the direction of Dr. J. H. Gibbon, Jr., to permit an evaluation of certain of the bronchologic aspects of this method of diagnosis. No reference will be made to studies of sputum for diagnostic purposes. This has been discussed in previous articles and was not employed in the series of cases included in this report.

Collection of Secretions.—A special collector equipped with large and small, straight and curved, flexible tipped aspirating tubes is employed to secure secretions.[2] Whenever possible secretions are collected from the lobar or segmental bronchi of the involved lung. Before completing the aspirations two to five cc of physiological saline solution are routinely instilled in this bronchus and reaspirated as a part of the specimen for examination.

There has been no essential change in the technic of smearing, fixing and staining the specimens or in the microscopic studies.

The number of specimens examined as of March 12, 1951 totalled 2814. No attempt has been made to determine the total

number of patients examined for in a certain number two, three or even more bronchoscopic procedures have been done and specimens of secretion secured for examination. Suffice to say that in positive cases the initial examination usually is found positive and repeated examinations are commonly made in the questionable or ultimately negative cases. In the group of 2814 specimens there were included 488 cases of bronchogenic carcinoma. The positive bronchoscopic findings and the results of the cytologic studies are listed under Table I. Of interest is the relatively low percentage rate of positive bronchoscopic biopsies (31.5 percent) and the high rate of positive cytologic diagnoses (56 percent) in the absence of a positive biopsy. In the absence of this finding a positive diagnosis would have necessitated an exploratory thoracotomy.

TABLE I.

Total number of examinations		2814	
Cases of Carcinoma		488	
Visual bronchoscopic evidence		309	
Positive bronchoscopic biopsy		154	(31.5%)
Positive cytologic findings		274	(56.0%)
Diagnosis made by biopsy	154—31.5%		
Diagnosis made by cytology	274—56.0%		
	87.5%		

In the bronchoscopic examination all visual evidence of suspected neoplasm is recorded. This includes deformity, fixity, rigidity, stenosis, infiltration or ulceration of any portion of the tracheobronchial tree and gross evidences of tumor within the bronchus. Secretions are secured for bacteriological and cytological study and a biopsy is done if any abnormal tissue is observed within the lumen of the bronchus. While deformity, fixity, rigidly, stenosis or infiltration of a bronchus cannot be accepted as positive evidence of neoplasm since it is observed in benign lesions as well, it nevertheless must be considered as suggestive of neoplasm and in conjunction with the roentgen findings and other evidences. A positive cytologic report is accepted as unequivocal evidence of carcinoma and in the same light as a positive biopsy. The presence of suspicious cells is not considered as sufficient evidence per se for surgical intervention but, as in the case of deformity, rigidity and similar finding it, too, can be considered as suggestive.

False positives or over-diagnoses apparently cannot be avoided, but the number is not great if one observes carefully certain criteria in the cytologic study. There have been no false positives in the

past four years. In a report concerning this[3] the following statement was made: "There have been only four over-diagnoses and these were in the earlier part of the studies. Three were cases of pulmonary abscess in which there was metaplasia of the epithlium. In each case there was only one nest of rather regular squamous cells. With added experience these offer no difficulty for since then we have picked up several similar cases which we did not call neoplastic. In one case a diagnosis of carcinoma was made by finding a single cell but on looking at that now it was not justified."

In our efforts to evaluate cytologic methods of diagnosis, the preoperative diagnostic findings were compared with the bronchoscopic biopsy rate, the roentgen findings, the presence of metastasis found at operation, and the rate of resectability.

In a previous report 69 consecutive cases of pneumonectomy performed by Dr. J. H. Gibbon, Jr., were studied, from the standpoint of diagnostic findings preoperatively and the pathological findings at operation to determine if there was any notable difference in cases cytologically positive and bronchoscopically negative as compared with cases cytologically and bronchoscopically positive.[2] In 18 cases with no visual bronchoscopic evidence of neoplasm and cytologically positive there was demonstrable evidence of metastasis to regional lymph nodes in only two. In 21 cases showing a positive bronchoscopic biopsy and positive cytologic findings there was metastasis in 11. In 23 cases which exhibited visual evidence of deformity, fixity and stenosis with no biopsy but with cytologic findings in 18 there was metastasis in 12. Of interest were seven cases with no metastasis in which both bronchoscopic and cytologic findings were completely negative.

It must be understood that any study of bronchoscopic diagnostic methods should not be on a competitive basis and, while a positive bronchoscopic biopsy should take precedence over a positive cytologic study, the thoracic surgeons now consider a positive cytologic diagnosis in the same light as a positive biopsy report. Visual evidence unsupported by a positive biopsy is considered only as a suggestive evidence as also is the findings of suspicious appearing cells. It has been our experience that in practically every case where a positive bronchoscopic biopsy was secured a positive cytologic study also was obtained. We therefore became interested in determining the results of a cytologic study of secretions where no tissue was available for biopsy.

In a series of 322 proven cases of bronchogenic carcinoma which were examined by bronchoscopy and cytology and evaluated by Dr.

John H. Gibbon, Jr., and his staff for surgical treatment, a study was made to determine the number of cases presenting visual bronchoscopic evidence, namely fixity, rigidity, deformity, ulceration or endobronchial neoplasm and the number in which a positive cytologic diagnosis was made in whom no biopsy could be secured. The findings are listed in Table II.

TABLE II.

Number of cases examined by Bronchoscopy and Cytology		322	
Visual bronchoscopic evidence		163	(50.6%)
Positive biopsy		81	(25.1%)
Positive cytology		174	(54.0%)
Diagnosis made by biopsy	81—25.1%		
Diagnosis made by cytology	174—54.0%		
	79.1%		

Although there was visual bronchoscopic evidence of disease in 50 percent of these it was possible to secure material for biopsy in but 25 percent, while neoplastic cells were found in an additional 174 cases or 54% of the total number. A tissue diagnosis therefore was made in 79.1 percent.

Of the 322 cases, 137 were resected. The visual bronchoscopic evidence, positive bronchoscopic biopsy and positive cytologic findings in those cases in whom no biopsy could be done is shown in Table III.

Curiously the biopsy rate in these was slightly higher than that obtained in the entire series and the positive cytologic findings were lower. This cannot be readily explained. Bronchoscopically accessible lesions usually are more often not resectable.

TABLE III.

Total number of resected cases		137	
Visual evidence		67	(48.9%)
Positive biopsy		38	(27.7%)
Positive cytology		68	(49.6%)
Diagnosis made by biopsy	38—27.7%		
Diagnosis made by cytology	68—49.6%		
	77.3%		

In the series of 137 resected cases a sample group of 63 in whom no gross evidence of metastasis to regional or distant lymph nodes could be demonstrated was studied. These were considered as cur-

ative pneumonectomies since the entire lesion appeared to have been removed surgically. This group is shown in Table IV.

TABLE IV

Curative Pneumonectomy (no evidence of metastasis)		63	
Visual evidence		31	(49.2%)
Positive biopsy		11	(17.4%)
Positive cytology		38	(60.3%)
Diagnosis made by biopsy	11—17.4%		
Diagnosis made by cytology	38—60.3%		
	77.7%		

Although the total percentage rate of tissue diagnosis in this group (77.7%) is almost equal to that of the entire series (79.1%) the disproportion between the bronchoscopic biopsy rate is striking and suggests that a high biopsy rate implies a low operable rate and a low biopsy rate a higher rate of operability. The higher cytology rate emphasized its importance diagnostically in lesions beyond the field of direct vision.

The nonresectable cases totalling 185 included 61 in whom an exploratory thoracotomy was done but resection was not completed and 99 cases that were obviously inoperable. The diagnostic findings in this group are listed as Table V.

In this group the tissue diagnostic rate of 80.5 percent varies little from that of the entire series of cases (79.1%) or even from the total rate for those considered most favorable for surgical treatment.

TABLE V.

All nonresected cases including 86 explored but not resected and 99 obviously inoperable cases		185	
Visual evidence		96	(51.9%)
Positive biopsy		43	(23.2%)
Positive cytology		106	(57.3%)
Diagnosis made by biopsy	43—23.2%		
Diagnosis made by cytology	106—57.3%		
	80.5%		

Of the 99 cases that were considered inoperable for obvious reasons the diagnostic data shown in Table VI indicate that this group gave the highest rate of preoperative diagnosis, namely 89.8 percent. The reason for this should be explainable on the basis of

more extensive disease and intrusion into the larger bronchi. The biopsy rate however, was not much higher than in several of the other categories.

TABLE VI.

Obviously inoperable cases		99	
Visual evidence		49	(49.4%)
Positive biopsy		25	(25.2%)
Positive cytology		64	(64.6%)
Diagnosis made by biopsy	25—25.2%		
Diagnosis made by cytology	64—64.6%		
	89.8%		

Additional interesting evidence to support the belief that cytologic diagnosis is an important aid was the finding of 8 cases, in the total series of 322 in which there was no roentgen evidence of bronchopulmonary disease, yet were diagnosed as bronchogenic carcinoma by bronchoscopic biopsy in two cases and positive cytology in six, a diagnostic rate of 100 percent.

It has not been possible to evaluate these diagnostic aids on the basis of five year survivals since the method of cytologic diagnosis was started slightly more than 5 years ago and too few cases are available at the present time. There are available, however, certain general data which may have some bearing on this, namely a comparison of operability rates. Thoracic surgeons generally hold to certain fundamental indications concerning the operability of bronchogenic carcinoma. It would appear reasonable therefore to compare the operability rates in large series of cases and possibly ascribe reasons for any outstanding variations. In the series of 322 cases considered in this report and treated by Dr. Gibbon or his staff the operability rate was over 41 percent. On the basis of reports available in the literature there is none that exceeds this.

Ochsner, DeBakey and Dixon[4] report a resection rate of 35.8 percent. All other reports available in medical literature are under 30 percent.

SUMMARY AND CONCLUSIONS

The findings by bronchoscopic biopsy and cytologic study of bronchoscopically removed secretions in the diagnosis of bronchogenic carcinoma have been presented and discussed from a practical clinical standpoint. While a positive biopsy report gives greater diagnostic security, a positive cytologic finding has been found equally dependable from a practical clinical standpoint. In prac-

tically every case of a positive biopsy the cytologic findings too were positive. The number of positive cytologic diagnoses in the cases of negative bronchoscopic biopsies were more than twice as great in nearly all groups. This supports the conclusion that cytologic study of secretions has added enormously in arriving at a positive preoperative diagnosis of carcinoma and has increased the rate of positive diagnosis in patients exhibiting negative bronchoscopic findings. It also has aided in the diagnosis of bronchogenic carcinoma in a small group of patients before there was any demonstrable roentgen evidence of bronchopulmonary disease.

128 South Tenth Street.

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DISCUSSION

Dr. John L. Pool (New York, N. Y.): Dr. Papanicolau in reporting on 403 proven cases of lung cancer obtained a positive smear in 252 or 54.4%. For the purpose of this discussion, I checked our figures at Memorial Hospital where cancer of the lung was re-sected. Our figures show that in 73 who had had preliminary sputum examination, 47, or 64%, were positive.

Another 46 patients were found on bronchial washings to be positive in 33, or 71%, which is a slightly better procedure, but one not so readily capable of repetition. I would recommend five sputum examinations in each case. In 27 patients, where both methods were employed, a positive smear for cancer cells was obtained in 22, or 81%. By contrast, bronchoscopic diagnosis was reached in only 38% of re-sected cases subjected to this diagnostic procedure.

Positive sputum, or positive washings, are a great benefit to the thoracic surgeon in planning his operative procedure. Absence of cancer cells in such smears requires at thoracotomy, further histologic examination by aspiration biopsy, incisional biopsy and frozen section or excisional biopsy, which delays the definite operative procedure.

In the follow-up care of pneumonectomized patients, study of the sputum aids in prognosis and in evaluating inter-current infections of the remaining lung. Finally, after x-ray and radium treatment of the non-resectable lung cancer, changes from positive to negative and vice versa, give valuable prognostic information.

LXXVI

CARINAL BIOPSY

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AND

COLEMAN RABIN, M.D. (By Invitation)

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The bronchoscopist has three functions in the diagnosis of malignancy of the bronchus. He must ascertain the presence of a lesion; he must obtain tissue by biopsy for histological examination and, by swab or aspiration, for the Papanicolaou smear; and he must determine the location and, if possible, the extent of the neoplasm.

The third function, the determination of the location and the extent of the growth, is of importance to the surgeon. This information helps in deciding on the operability when cure of the disease is possible. It is undoubtedly true that in most instances the bronchoscopic findings are not determinants of a curative operation. In the great majority of instances, invasion of the mediastinum, of the lymph nodes or of the pleural space are the limiting factors and the bronchoscopist can offer little information on these involvements. Nevertheless, when the bronchoscopist finds that a growth extends from the bronchus to invade the tracheal wall or that it crosses over to the opposite main bronchus, he has diagnosed a lesion incurable by simple pneumonectomy. Furthermore, rigid pressure on the medial bronchial wall or broadening of the carina indicates mediastinal extension of the growth or lymph node involvement and therefore an inoperable condition. These findings are not too common and are generally associated with obvious findings of advanced disease so that the bronchoscopic signs are merely corroborative.

We have found that a biopsy of the carinal region is positive in a good many cases of large branch bronchus carcinoma. By large branch bronchus, we mean the upper, middle and lower lobe bronchi and their primary divisions.

The specimen is taken from the following three places: the edge of the carina by gently stripping off the mucosa; from the mucosa of the upper one to two centimeters of the medial wall of the main

bronchus below the carina; and from the lateral wall of the main bronchus at the level of the carina. Grossly the mucosa may appear normal but in some cases one gets the impression of thickening of the mucosa. In any case, a specimen is taken from the carinal region as described above when operation is contemplated. Positive carinal biopsy shows lymphatic or submucosal invasion by carcinoma.

A positive carinal biopsy signfies an inoperable lesion from the standpoint of possible cure. Inasmuch as we are dealing with a lymphatic invasion or extension, resection beyond the limits of disease is impossible. On the other hand, a negative carinal biopsy merely indicates absence of disease by invasion in the carinal region. The surgeon can therefore plan his resection within one to two centimeters of the carina with the knowledge that no disease is present in this mucosal region. A negative carinal biopsy yields no information about the other regions where invasion may occur and as a matter of fact, a great many patients with negative carinal biopsies are inoperable because of mediastinal or lymph node invasion.

It is suggested that the carinal biopsy be performed routinely before deciding on the surgical treatment of carcinomas of the bronchi. In the two hospitals in which our work has been done, the value of the procedure has been recognized for the past seven years to the extent that the clinicians and surgeons dealing with thoracic diseases have requested carinal biopsy before thoracic exploration.

The demonstration of the carinal invasion via the lymphatics is important also because of the possibility of cure in these patients as revealed by recent work. Dr. William Harris, radiotherapeutist to The Mount Sinai Hospital, N. Y., is using a new technic which has eradicated the carinal and bronchial disease in some patients so that curative surgery could be performed. Bronchoscopy and pneumonectomy after radiotherapy demonstrated the eradication of the invasion.

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LXXVII

ENDOBRONCHIAL LYMPHOMA AND ITS SIMULATION BY BRONCHOGENIC CARCINOMA

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AND

RALPH JONES, JR., M.D. (By Invitation)

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The intrathoracic manifestations of the malignant lymphomata are of particular interest because of the vital structures affected, and because of the close similarity of these lesions to the more common pulmonary diseases. The frequency with which these lymphomatous diseases are accompanied by intrathoracic involvement is well known, and the specific nature of the underlying pathologic process is usually recognized. However, occasionally the clinical manifestations of the malignant lymphomata are confined to the chest, particularly in Hodgkin's disease; it is in such cases that misdiagnoses occur.

The incidence and sites of involvement of intrathoracic Hodgkin's disease are shown in Table I, a summary of information adapted from Vieta and Craver.[1] In the chest, Hodgkin's disease may involve lymph nodes, pulmonary parenchyma, and the tracheo-bronchial tree. As would be expected, the incidence of all the various lesions at postmortem examination was considerably greater than on routine roentgenogram of the chest. Mediastinal and hilar lymph node enlargement was the most common lesion, but parenchymal involvement was frequently demonstrated. It is probable that pulmonary tissue is the most frequent site of Hodgkin's disease, second only to lymph node involvement.[1, 2] Indeed, it is held by Versé that pulmonary tissue is the *primary* site of origin of Hodgkin's disease in a significant number of cases.[3] Although intrabronchial lesions were found in a surprisingly large number of cases, this was the predom-

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TABLE I.
INTRATHORACIC LESIONS IN HODGKIN'S DISEASE*

	No. of Cases	Mediastinal & Hilar Node Enlargement (percent)	Intrabronchial Spread (percent)	Parenchymal Lesions (percent)	No Chest Lesions (percent)
Roentgenographic Findings	335	58.5	.....	38.5	25.3
Autopsy Findings	51	74.0	13.7	47.0	12.0

*Adapted from Vieta and Craver.

inant lesion in only one instance. Nevertheless, it is apparent that a significant degree of bronchial involvement is not an uncommon feature of this disease.

The matrix for the endobronchial lesion is the lymphoid tissue which is found in both diffuse and in follicular aggregates in the mucosa and in the fibrous tissue surrounding the cartilage, while lymph nodes are present along the larger bronchi. The endobronchial lesion of Hodgkin's disease is commonly a granulomatous infiltration of the mucosa, producing plaque-like opacities or elevated areas which may ulcerate with continued growth. These ulcerated, nodular growths may be so numerous that they almost completely cover the inner surface of the trachea and large bronchi.[4] The specific nature of the disease is suggested by its multiplicity and bilaterally, aiding in the distinction of Hodgkin's disease from carcinoma, the lesions of which typically tend to be single or closely grouped. Single polypoid or adenomatous-appearing masses projecting into the bronchus and narrowing the lumen are rare in Hodgkin's disease.[5] Displacement of tracheo-bronchial structures is an infrequent occurrence in this disease, but extramural compression by enlarged lymph nodes is well recognized.[6]

The broncho-pulmonary lesions of Hodgkin's disease are usually associated with manifestations of the disease elsewhere in the body, but it is important to emphasize that the predominant, and occasionally the *only* lesions may be restricted to the lungs, and there may be no coincident or antecedent peripheral adenopathy. Case reports 1 and 2 illustrate well the problems that may be presented when this occurs.

CASE 1.—M. R., a 45 year old white female, developed a small mass in the supraclavicular area in 1946. This was biopsied and revealed only non-specific lymphadenitis. She was well until June, 1948, when she noted shortness of

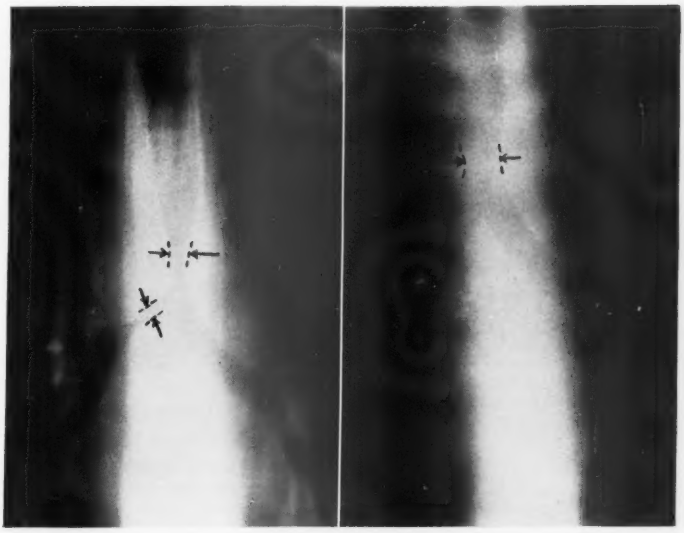

Fig. 1, Case 1.—June 24, 1948. Body section film before therapy.

Fig. 2, Case 1.—August 10, 1948. Body section film following roentgentherapy.

breath and persistent cough. On admission to the University of Pennsylvania Hospital, physical examination was entirely negative except for bilateral rhonchi and wheezes. No peripheral adenopathy was present, and the liver and spleen were not palpable. Roentgenogram of the chest revealed mediastinal widening. A body section film (Fig. 1) showed constriction of the lower trachea and the right main bronchus. At bronchoscopy, a fungating growth with the appearance of a malignant tumor was found to be narrowing the lumen of the trachea and extending into the right main bronchus, and to a lesser extent into the left main bronchus (Fig. 5*A*). The bronchial biopsy (Fig. 6) presented a histologic picture compatible with Hodgkin's disease.

The patient was given 2 courses of roentgen therapy through anterior and posterior mediastinal portals, receiving a total tissue dosage of 3720 roentgens. Almost complete alleviation of her symptoms occurred, and a body section film following the course of therapy showed marked improvement in the tracheobronchial lesions (Fig. 2). Three months later symptoms recurred, and a second bronchoscopy revealed an ulcerated growth confined to the right main bronchus with narrowing of the lumen at the bifurcation (Fig. 5*B*). The patient was then treated with intravenous nitrogen mustard, receiving 5.3 mgm. of this material on three successive days, followed by a third course of roentgen therapy to the anterior and posterior mediastinal regions, bringing the total dosage of the latter to 6800 tissue roentgens. She remained essentially asymptomatic for two years (November 1950) when recurrence developed. Again her symptoms were entirely respiratory in nature and all evidences of Hodgkin's disease were restricted to the chest. She presented the picture of acute respiratory obstruction with rather marked obstructive emphysema of the left lung. A body section film

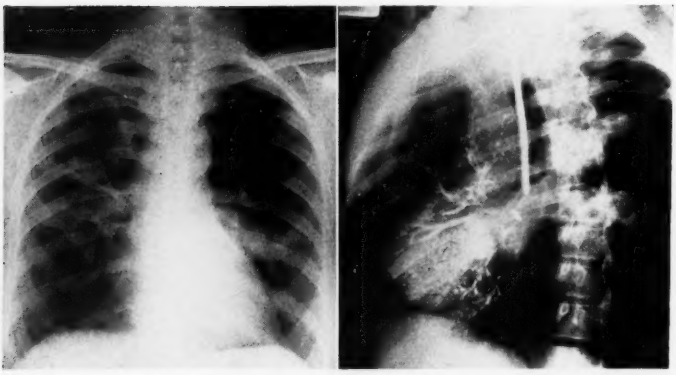

Fig. 3, Case 2.—February 18, 1950. Initial roentgenograms of the chest.

Fig. 4, Case 2.—March 30, 1951. Bronchograms showing abnormal filling of the bronchus to the apical segment of the left lower lobe.

showed lower tracheal and bilateral bronchial narrowing. At bronchoscopy the process was considerably more extensive than before (Fig. 6C). Ulcerated, fungating lesions extended down the trachea circumferentially from one inch below the level of the larynx into the left main bronchus to the orifice of the left upper lobe bronchus. The left main stem bronchus was almost completely obstructed by granulomatous tissue. This structure was literally "cored-out," and aeration of the emphysematous lung was greatly improved. The main bronchus was appreciably narrowed by a cicatrix. The patient received a second course of nitrogen mustard therapy, consisting of a total of 30 mgm given over a three day period, and at present the disease is quiescent.

It is important to note that at no time during the three-year period in which the patient was followed were there apparent manifestations of Hodgkin's disease outside the chest. Significant adenopathy was not observed and enlargement of the liver or spleen was not noted.

Case 2.—L. D., a 39 year old white female, developed persistent cough and weakness in December, 1948, and was found to have generalized lymphadenopathy. Roentgenograms of the chest showed mediastinal widening. Biopsy of a supraclavicular node presented an histologic picture typical of Hodgkin's disease. Roentgen therapy to the involved areas produced remission of all subjective and objective evidence of disease. In February, 1950, there was gradual recurrence of her symptoms, and cough with blood-streaked sputum was noted for the first time. She was admitted to the University of Pennsylvania Hospital at this time, and physical examination revealed a moderate peripheral lymphadenopathy and hepato-splenomegaly. Roentgenogram of the chest (Fig. 3) showed hilar adenopathy and bilateral parenchymal infiltrations. She was treated with intravenous nitrogen mustard, receiving 6 mgm of this material on four successive days, and all evidence of her disease disappeared. The disease was quiescent until Novem-

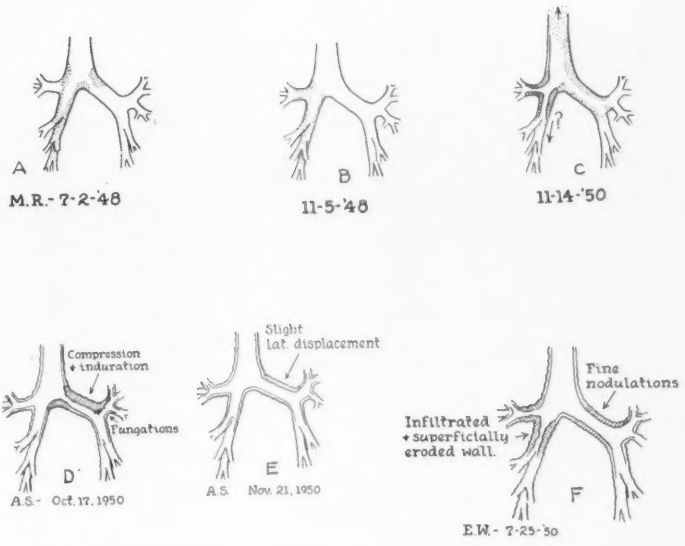

Fig. 5.—Diagramatic representation of bronchoscopic findings.

ber, 1950, when exacerbation occurred. Oral triethylene melamine therapy produced only partial relief of her symptoms. Fifty-five mgm of this drug was given in 5 to 10 mgm doses at weekly intervals over a three month period. The chronic cough was refractory to therapy and became productive of increasing amounts of blood-streaked sputum, and eventually frank hemoptysis occurred. Tuberculin tests (.005 mgm of P.P.D.) were negative and repeated sputum examination failed to show acid-fast bacilli. No lesion was demonstrated on bronchoscopy on two occasions. In March, 1951, bronchoscopy was repeated, and blood was observed exuding from the left bronchus, but the source of the bleeding was not identified. A bronchogram (Fig. 4) showed abnormal filling of the bronchus to the apical segment of the left lower lobe, suggesting an endobronchial lesion beyond the limits of vision through the bronchoscope. Roentgen therapy was administered to this site through small anterior and posterior portals, the patient receiving a total air dosage of 1100 roentgens. Her persistent cough and hemoptysis were promptly relieved with this therapy.

COMMENT

The first patient has had a recurrent endobronchial lesion over a period of three years, which on repeated biopsy showed the histologic picture of Hodgkin's disease. On only one occasion was a peripheral lymph node sufficiently prominent to warrant biopsy, and this node showed no evidence of Hodgkin's disease. The response to the various types of therapy has always been typical of that seen with Hodgkin's disease. Each time the disease recurred in the bronchus, the patient was faced with imminent danger of complete res-

TABLE II.
INTRATHORACIC LESIONS IN THE LYMPHOMATA
(AUTOPSY FINDINGS)*

Disease	No. of Cases	Mediastinal & Hilar Node Enlargement (percent)	Intrabronchial Spread (percent)	Parenchymal Lesions (percent)	No Chest Lesions (percent)
Lymphosarcoma	55	64.7	.....	24.5	27.0
Lymphatic Leucemia	31	83.0	3.0	19.0	16.0
Myelogenous Leucemia	23	47.0	.....	13.0	52.0

*Adopted from Vieta and Craver.

piratory obstruction; if her life is not abruptly terminated by such a catastrophy the disease may well eventually become disseminated.

The second case, on the other hand, was generalized at the outset and all evidences of the disease responded to systemic therapy, except the resistent endobronchial lesion. For the past seven months the lungs have been the only apparent site of her disease, and cough and hemoptysis the only symptoms. Because of the rarity of hemoptysis in Hodgkin's disease, repeated unsuccessful attempts were made to explain it on the basis of a co-existent pathologic process (i.e., bronchiectasis, pulmonary tuberculosis). However, the demonstration of a bronchial abnormality on bronchogram, together with a prompt response in her symptoms following roentgen therapy suggests a lesion of Hodgkin's disease involving the bronchus.

In the malignant lymphomata other than Hodgkin's disease (Table II), enlargement of mediastinal nodes and parenchymal lesions are common, but clinically significant endobronchial lesions have not been reported. In lymphosarcoma (giant follicle, lymphocytic and reticular cell types), large mediastinal masses may be present. These masses rarely invade tracheobronchial structures; rather they tend to envelope them. According to Vieta and Craver,[1] "retraction of the lung or displacement of the trachea so common in cases of carcinoma of the lung, is rarely seen in lymphosarcoma." Our experience has not been in agreement with this opinion. During the past year we have observed three cases of lymphosarcoma in which displacement of intrathoracic structures was a prominent feature.

In lymphatic leukemia, mediastinal and hilar lymph node enlargement is the most frequent type of intrathoracic lesion found

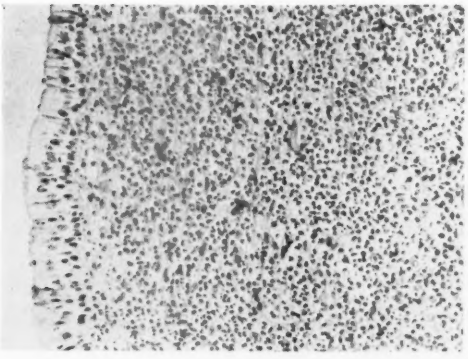

Fig. 6, Case 1.—Endobronchial biopsy: Hodgkin's disease (x200).

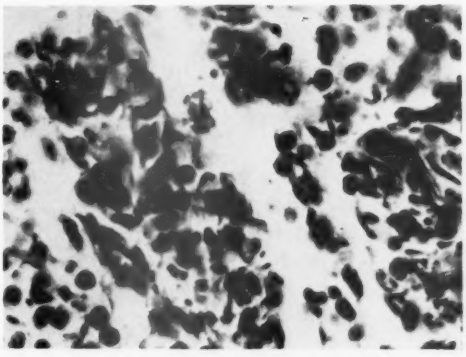

Fig. 7, Case 3.—Endobronchial biopsy: anaplastic carcinoma indistinguishable from malignant lymphoma (x1020).

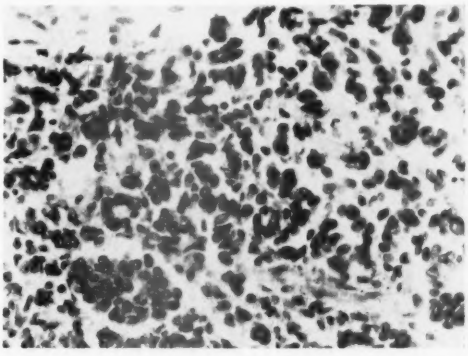

Fig. 8, Case 3.—Postmortem section of the left lower lobe bronchus: undifferentiated bronchial carcinoma (x450).

at postmortem examination. Symptoms due to tracheo-bronchial compression are not infrequently produced, and may be increased by the acute swelling attending roentgen therapy. Although infiltration of nodes by myeloid cells was present at autopsy in 47 percent of the cases of myelogenous leukemia (Table II), roentgenograms of the chest failed to reveal significant adenopathy in a single case.[1] This is in keeping with the pathologic finding that greatly enlarged nodes in this disease are uncommon, and respiratory symptoms due to compression or direct invasion of pulmonary tissues are decidedly rare. Thus, of the various lymphomatous processes that produce intrathoracic lesions, only in Hodgkin's disease have clinically significant endobronchial lesions been found.

The diagnosis of a malignant lymphoma restricted to the broncho-pulmonary tree is often uncertain. The unusual character of the case, and the difficulty of obtaining sufficient biopsy material are the chief problems. Moreover, confusion with inflammatory diseases and other neoplastic lesions of the lung provide the main pitfalls in diagnosis. Many authors agree that there are no specific radiologic features of Hodgkin's disease, and the radiologist at best can only make a presumptive diagnosis of a malignant lymphoma. In the case of a middle aged individual with Hodgkin's disease presenting a combination of hilar and endobronchial lesions without other manifestations, it is virtually impossible to differentiate clinically this disease from bronchial carcinoma. Papanicolaou smears are of little avail in distinguishing between these neoplasms,[17] and biopsies are often inconclusive (see case reports 3 and 4). The response to roentgen as well as to nitrogen mustard therapy may be as dramatic initially in bronchial carcinoma as in the lymphomata (cases 3 and 4). In such a patient with Hodgkin's disease a mistaken diagnosis of bronchial carcinoma may well be made, and only the clinical course of the patient and the mode of eventual dissemination of the disease will establish the true diagnosis.

Conversely, bronchial carcinoma may masquerade as an intrathoracic lymphoma.[13, 18, 20] This fact has not been sufficiently emphasized in the literature. The structural versatility of the tumor and its vagaries of metastatic spread, often produce a clinical picture that may readily be mistaken for a lymphoma. The combination of bronchial or hilar lesions, together with axillary or cervical adenopathy is a common clinical picture in anaplastic bronchial carcinoma (see cases 3 and 5), and may not infrequently also be observed in the malignant lymphomata. Ochsner[19] found spread to these peripheral sites in 28 percent of his series of 1298 cases of carcinoma of the lung. In such cases, histologic examination of the involved cer-

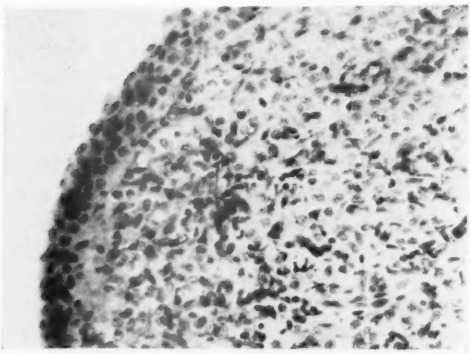

Fig. 9, Case 4.—Endobronchial biopsy: pleomorphic bronchial carcinoma resembling and initially diagnosed lymphosarcoma (x450).

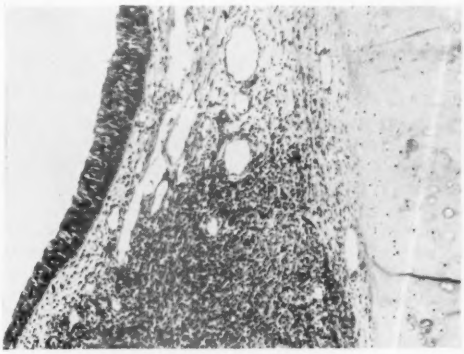

Fig. 10, Case 4.—Postmortem section of the left main bronchus; anaplastic bronchial carcinoma (x120).

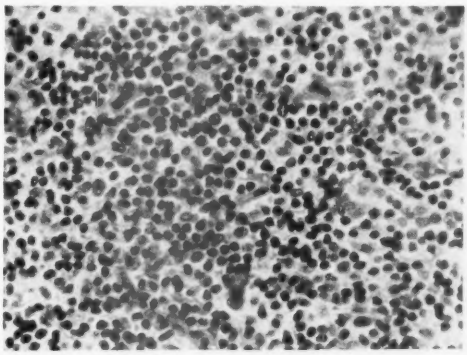

Fig. 11, Case 5.—Lymph node biopsy: metastatic bronchial carcinoma misdiagnosed as malignant lymphoma of undetermined type (x 640).

vical or axillary node often perplexes and elicits differences of opinion from experienced pathologists, particularly when the tumor is of the anaplastic type. There is no greater bane to the surgical pathologist as well as to the clinician than the "doubtful lymph node." Malignant lymphomata, inflammatory reactions, reticulo-endothelial hyperplasia and pleomorphic anaplastic carcinoma are difficult and occasionally impossible to differentiate on the basis of the histologic picture present in a lymph node.[18] It is aptly stated by Willis[11] that, "no pathologist lives long enough to be infallible in this field." The bronchologist is of inestimable value in this situation, but likewise may be confronted with a dilemma. Bronchoscopic biopsy material may often show an histologic picture compatible with either pleomorphic, undiffereniated carcinoma or malignant lymphoma. Finally, careful necropsy with particular attention to gross as well as microscopic aspects may be the only means of distinguishing between these neoplasms.

Recently, we have observed three cases (3, 4, and 5) in which the initial diagnosis was held to be Hodgkin's disease or other lymphoma, but which subsequently proved to be anaplastic bronchial carcinoma.

REPORT OF CASES

CASE 3.—A. S., a 44 year old white male, was well until September, 1950, when he developed a dry, hacking cough associated with increasing shortness of breath, weakness and anorexia. Enlarged bilateral supraclavicular nodes were present, diagnosed at biopsy, "malignant lymphoma." A mediastinal mass was present in the roentgenograms of the chest, and the patient was treated with roentgen therapy through anterior and posterior mediastinal portals, receiving a total air dosage of 4500 roentgens. There was prompt but transient improvement of his symptoms. When first seen at the University of Pennsylvania Hospital in October, 1950, the patient was acutely ill with physical signs of decreased aeration of the left lung. There were enlarged, firm supraclavicular nodes on the left. No other positive physical findings were observed. Roentgenogram of the chest showed mediastinal enlargement. A body section film revealed a distortion of the inferior aspect of the left main bronchus. At bronchoscopy, the left main bronchus was more horizontally placed than normal, and there was an ulcerated, fungating lesion at the bronchial opening to the left upper lobe (Fig. 5*D*). Histologic study (Fig. 7) of material obtained at bronchoscopy was suggestive of either carcinoma or malignant lymphoma of undetermined type.

The patient was treated with intravenous nitrogen mustard, receiving 50 mgm of this material over a period of two weeks, and within ten days was entirely asymptomatic. At this time, a body section film revealed no evidence of encroachment on the left main bronchus, and at bronchoscopy (Fig. 5*E*) there was no evidence of the lesion previously described. The only abnormality noted was slight lateral displacement of the left main bronchus. The patient was symptom free for approximately two months, then developed progressive respiratory involvement and superior vena canal obstruction. At this time a roentgenogram of the chest showed an extensive process involving both lungs and the mediastinum with marked constriction of the trachea. The patient was refractory to further treatment and rapidly succumbed.

At autopsy, an extensive bronchial carcinoma was found, apparently arising from the inferior aspect of the left main bronchus with constriction of the branch bronchi and extension into the adjacent lung parenchyma. There was marked mediastinal and paratracheal involvement, greatly reducing the lumen of the latter structure. Widespread, early metastases were present in the liver, pancreas, adrenals and other sites of spread typical of bronchial carcinoma. The histologic picture (Fig. 8) was characteristic of a very anaplastic, pleomorphic carcinoma with little tendency to alveolar formation.

CASE 4.—E. W., a 25 year old white female, entered the University of Pennsylvania Hospital in July, 1950, after a three month illness characterized by progressive dyspnea, cough, weakness and weight loss. Physical examination was essentially negative except for the presence of bilateral rhonchi and wheezes. Roentgenogram of the chest revealed mediastinal and left hilar masses. At bronchoscopy, the right main bronchus was narrowed and its wall superficially infiltrated and eroded (Fig. 5*F*). A similar process was present on the left side with less narrowing of the lumen and fine nodulations extending throughout the mucosa. Endoscopic biopsy of this lesion (Fig. 9) showed an histologic picture compatible with either reticulum cell lymphosarcoma or Hodgkin's disease.

The patient received intensive roentgen therapy through large anterior and posterior mediastinal portals, to a total tissue dosage of 2100 roentgens. There was prompt and dramatic improvement of her symptoms, but no appreciable change was demonstrated in serial roentgenograms of the chest. Four months later, the patient died suddenly at home, having had no apparent difficulty since her previous hospitalization except for residual weakness and continued weight loss.

At autopsy, an anaplastic bronchial carcinoma was found arising from the left main bronchus (Fig. 10) with widespread visceral metastases. No evidence of a malignant lymphoma was found. The sudden death was explained by the fact that eighty percent of the myocardium was replaced by tumor.

CASE 5.—J. K., a 44 year old white male, entered the University of Pennsylvania Hospital in August, 1950, for treatment of advanced arteriosclerotic vascular disease of the right lower extremity associated with diabetes mellitus. Five weeks after mid-thigh amputation of the gangrenous limb and while convalescing in the hospital, he developed a generalized lymphadenopathy and an enlargement of the liver which rapidly extended to the iliac crest. At this time, the total white blood count was 30,000 with 30% immature forms: 8% myeloblasts, 14% promyelocytes, 8% myelocytes, together with 6% nucleated red blood cells. Study of the bone marrow suggested a leukemoid reaction, but the possibility of leukemia could not be unequivocally excluded. An axillary lymph node was removed (Fig. 11) the architecture of which was completely replaced by sheets of dark-staining cells not unlike adult lymphocytes. An histologic diagnosis of lymphoma of undetermined type was made. This picture persisted for several weeks until rapidly progressing respiratory symptoms developed. Roentgenogram of the chest showed mediastinal widening. At bronchoscopy many of the branch bronchi were plugged with inspissated secretions (Fig. 5*G*). There was blunting of the carina, thickening of the right main bronchus and deformity of the right upper lobe bronchus. Endobronchial biopsy material failed to reveal the underlying pathologic process. The patient died four days later, two months after the onset of his disease, due to progressive respiratory obstruction.

At postmortem examination, a bronchial carcinoma was present arising from the right upper lobe bronchus with widespread metastases.

In each of these three cases of bronchial carcinoma, an initial diagnosis of malignant lymphoma was made on the basis of histologic study, and the clinical picture was compatible with the diagnosis in each case. In Case 3, the intrathoracic lesions were accompanied by enlarged supraclavicular nodes. The histologic picture of the lymph node biopsy was indistinguishable from a lymphoma. The response to treatment with roentgen and nitrogen mustard therapy was as dramatic as that which occurs when a malignant lymphoma is treated with these agents.

In Case 5, the leukemoid reaction produced by the extensive bone marrow invasion, together with the rapid development of the generalized adenopathy and the tremendous hepatomegaly while the patient was under observation in the hospital—all provide a picture well compatible with a malignant lymphoma. Moreover, the patient showed no respiratory symptoms until a few days before death. The true nature of the basic pathologic process was totally unsuspected until necropsy.

Metastasis by the blood stream occurs in bronchial carcinoma in three-quarters of fatal cases, with demonstrable skeletal metastases in 25 percent.[11] Extensive infiltration of the bone marrow by malignant cells may lead to blood abnormalities such as progressive anemia, leukemoid reactions[21] and purpuric states, presenting diagnostic problems that closely resemble those of malignant lymphomata.

The excellent response in Case 4 to roentgen therapy and the age (25 years) and sex of the patient were the prime factors which clinically suggested the diagnosis of lymphoma. Again, the bronchoscopic biopsy was inconclusive and more closely resembled lymphosarcoma. The finding of a bronchial carcinoma with a rather typical metastatic spread was a complete surprise at necropsy.

Clinical misdiagnosis in cases of bronchial carcinoma are still being made with great frequency, despite modern diagnostic methods such as radiography and bronchoscopy. Between 1931 and 1944, Willis[11] performed 84 necropsies on cases of bronchial carcinoma; of these 35 (42 percent) had been misdiagnosed clinically, 19 as another type of malignant disease and 16 as non-neoplastic disease. An accurate knowledge of the methods and routes of spread of bronchial carcinoma is of great value in establishing a diagnosis. Probably as many, if not more errors in diagnosis due to bizarre

metastases are made in this disease than in any other neoplastic disease.[11]

SUMMARY AND CONCLUSIONS

Intrathoracic lesions are common in the malignant lymphomata, particularly in Hodgkin's disease. In most cases, peripheral manifestations of the underlying disease are present, permitting accurate histologic diagnosis. Endobronchial involvement represents 14% of the intrathoracic lesions of Hodgkin's disease found at autopsy, but such lesions are rare in the other lymphomata.

Occasionally the only apparent lesions of Hodgkin's disease are confined to the broncho-pulmonary tree, presenting a clinical picture that may well be indistinguishable from bronchial carcinoma and other common pulmonary diseases. Two such cases are reported, and the problems of diagnosis and therapy are discussed.

Bronchial carcinoma, on the other hand, may often produce a clinical picture that masquerades as a malignant lymphoma. Endobronchial and peripheral lymph node biopsies may be inconclusive and the response to nitrogen mustard and roentgen therapy can be favorable in either disease. Three cases are presented which were originally diagnosed as malignant lymphoma, but which subsequently were proven to be bronchial carcinoma.

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DISCUSSION

DR. EDWARD B. BENEDICT (Boston, Mass.): The excellent paper by Doctor Atkins, Jones and Sullivan interests me very much. I have not recognized the multiple placque-like opacities in the main bronchi but I shall look for them in the future.

My own bronchoscopic experience in lymphoma concerns twelve cases in which the disease was proved histologically by lymph gland biopsy (7 cases), autopsy (2 cases), chest wall biopsy (1 case), peritoneoscopy with liver biopsy (1 case), and lobectomy (1 case). In only two cases was there evidence of intrinsic lymphomatous disease in the bronchial lumen; unfortunately the bronchoscopic biopsy in one of these cases was erroneously reported as undifferentiated carcinoma, and in the other case the bronchoscopic biopsy was too poorly preserved for diagnosis. In a third case biopsy obtained from the right lower lobe bronchus was reported squamous cell carcinoma grade III; in this case the axillary gland biopsy was malignant lymphoma (lymphocytic type); in other words that patient was unfortunate enough to have two types of malignant disease at the same time.

In two other cases bronchoscopy was entirely negative. In one of these a successful right upper lobectomy for malignant lymphoma (lymphocytic type), was carried out by Dr. E. D. Churchill. In the other one pleural effusion and axillary gland lymphoma made x-ray therapy the treatment of choice.

In the seven other cases bronchoscopy disclosed one or more of the following: wide, fixed, distorted carina; bronchi deviated, fixed, angulated and narrowed; bronchial obstruction from extrinsic pressure; paralyzed vocal cord. In many cases the bronchoscopist thought the findings were due to advanced inoperable carcinoma.

In my opinion lymphoma is almost never primary in the bronchus and usually involves the tracheobronchial tree only by extrinsic pressure. As previously stated "bronchoscopy in suspected or proven lymphoma is important (1) for the negative evidence it may give of an uninvolved tracheobronchial tree, thus permitting surgery; (2) because of the rare intrinsic bronchial invasion; (3) for the positive evidence it may give of widening and fixation of the carina or deviation, fixation, and narrowing of parts of the trachea or bronchi by extrinsic

pressure and (4) for the disclosure of a bronchogenic carcinoma or other intrabronchial pathology in addition to the lymphoma."

ROBERT D. SULLIVAN, M.D. (Philadelphia): Very briefly, I would like to state that despite the modern diagnostic facilities we have available, we are still missing a diagnosis of bronchogenic carcinoma. Willis, in 84 very careful and complete necropsies, which were done between 1931 and 1944, found that the diagnosis was missed in 35 cases, or 42 per cent. Usually, the diagnosis which was mistakenly made was that of some other neoplasm. In this situation, Papanicolaou smears are not helpful.

In 19 cases of these 35 which were missed, the diagnosis of an inflammatory situation was made. So that it certainly should be emphasized, and I know this group appreciates the fact, that we are not too skillful in making a diagnosis of bronchogenic carcinoma even today.

LXXVIII

TRACHEAL RESECTION WITH PRIMARY ANASTOMOSIS

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Lesions of the trachea, though not common, present a challenge to surgical management. Primary suture of the divided trachea following injury is successful if the tracheal edges are carefully coapted by individual suture technique. Strictures of the trachea though rare today were relatively common following diphtheria. Reports[1, 2, 3] published around the turn of the century describe excision of such strictures and primary end-to-end anastomosis. Complications resulted when the defect was over several tracheal rings.

Considerable experimentation on tracheal and tracheobronchial reconstruction has recently been performed. Traffel[4] in 1940 demonstrated that fascial transplants could be used to close small tracheal defects. These grafts did not survive but did provide the framework for fibroblastic infiltration. Some were shown to be covered with one layer of respiratory epithelium.

Daniel[5] (1948) attempted to solve this problem experimentally by bridging tracheal defects with rigid tubes of either glass, stainless steel, or Vitallium. He was able to demonstrate the development of segments of cartilage from fibroblasts within the granulation tissue surrounding these tubes in dogs sacrificed two weeks to six months postoperatively. Epithelial regeneration over the fibrous and granulation tissue, however, appeared delayed and in no instance was it complete. Lucite tubes have been employed experimentally by Davies[6] and in one case clinically by Longmire[7] to bridge tracheal defects. Jarvis[8] (1950) described the clinical use of a stainless steel tube to bridge a defect in the trachea following resection of a malignant cylindroma. This tube functioned well for one year at which time it was removed in the hopes that sufficient tracheal regeneration had developed. However, because of insufficient tracheal rigidity during coughing it was necessary to replace the tube within three weeks. This patient subsequently died from metastasis thirty-three months after the initial operation. Holmes[9] (1950) described the use of a Vitallium tube three-fourths inches in length to bridge a defect following excision of a traumatic stricture. Because of redundant tracheal mu-

cosa developing at each end this was replaced with a stainless steel tube.

Rob and Bateman[10] (1949) described the use of tatalum gauze covered with fascia to effect a repair of the cervical trachea following resection for a carcinoma of the thyroid. Burnett[11] (1950) described the use of wire mesh experimentally. Clagett[12] and his associates employed polythene tubes flanged at each end and in the middle to bridge such defects. Though most of the dogs did well the tubes in some became loose or dislodged.

The initial success with the use of rigid tubes was encouraging, however, the majority of such investigations were relatively short term experiments. The usual complications associated with the use of foreign bodies and the absence of tracheal rigidity following the removal of the tubes have prevented their widespread clinical application.

To obviate this criticism both autogenous and homogenous tracheal grafts have been employed experimentally with varying degrees of success by Carter and Strieder[13] (1950) and others.[14, 15] Unfortunately, again there has been insufficient time to allow for long term results. The high incidence of absorption of the grafts, particularly the homografts and preserved grafts and the subsequent development of stenosis has discouraged their clinical use even though promising early results were obtained with the use of autografts.

Pedicled skin grafts were employed by König[16] in 1896 and Trnka[17] in 1900. In the latter instance the graft was stiffened temporarily by needles. More recently Gebauer[18, 19] employed wire-enforced dermal grafts to enlarge the tracheal or bronchial lumen at the site of a tuberculous stricture. In such instances, a linear incision is made through the posterior membrane at the site of the stricture, the cartilagenous rings are spread apart and the residual defect covered by an inlay dermal graft. The procedure appears very satisfactory in the enlargement of strictures but may not be feasible to bridge gaps as would occur following complete tracheal resection for tumors where tension would be present.

Most of the above investigators have employed a variety of procedures in an attempt to solve this most difficult surgical situation. For the most part the greatest degree of success and the fewest complications have followed tracheal resection and primary anastomosis without the use of tubes or grafts. Ferguson[20] and his associates feel that as much as one-fourth to one-third of the tracheal length can be resected experimentally with end-to-end anastomosis. They stress the necessity of accurate apposition of the mucosal edges. This

method in their hands was superior to the use of grafts and various types of tubes. Lately Grindlay[21] and his associates as well as Juvenelle and Citret[22] have reported primary anastomosis of the left bronchus to the trachea without the use of prosthesis following resection of the distal trachea experimentally. It appears from the evidence so far attained that the results from primary anastomosis are superior to the use of various prosthesis and grafts and that there are fewer complications. As a temporary expedient in patients with advanced carcinoma rigid tubes may provide an adequate airway when necessary to bridge longer defects than what might be possible by primary anastomosis.

During the past two years five patients have had resections of portions of the trachea. One patient had a leiomyoma of the upper trachea causing obstruction and the other four had secondary involvement of the distal trachea from carcinomas arising in the right main stem bronchus. The surgical management of these patients will be discussed.

The patient with the leiomyoma was a 33-year old female who had evidence of increasing respiratory obstruction of six months duration manifesting itself in a marked inspiratory-expiratory wheeze. During the early course of her illness, the obstruction suggested the presence of intractable asthma. This required emergency hospitalization on two occasions and oxygen therapy. It soon became apparent that some other cause of the increasing respiratory distress was present and the patient was referred for a bronchoscopic examination. Roentgen examination of the chest was negative. Good visualization of the trachea was not possible by indirect laryngoscopy.

As the bronchoscope was introduced between the vocal cords, a large tumor was visualized arising posteriorly. This occluded at least three-fourths of the tracheal lumen. Endoscopic removal of the tumor was undertaken to provide an adequate airway. The base of the tumor appeared to extend 2 cm in length. The microscopical examination of this tumor revealed it to be a leiomyoma. It was obvious that the tumor would soon recur unless completely removed. Consequently, operation was recommended. Investigation performed on postmortem material revealed that sufficient tracheal mobilization could be obtained by freeing the trachea in the superior mediastinum to allow approximation and primary anastomosis of the divided tracheal ends following removal of four to five tracheal rings. Further mobilization could be obtained if necessary by dividing the sternum and freeing the distal trachea and main stem bronchi.

The operative resection required removal of the upper four tracheal rings and posterior third of the cricoid cartilage. The distal

trachea was then mobilized in the superior mediastinum by finger dissection. The divided ends were approximated and primary anastomosis performed with individual 0000 Deknatal suture. Care was exercised in placing the sutures to include mainly the fibroelastic tissue and a minimal amount of cartilage and mucous membrane. In several places it was necessary to include a portion of the cartilaginous ring and cricoid cartilage. A distal trachotomy was then performed and the incision closed. Care was exercised postoperatively to keep the chin in the neutral position to avoid undue tension. The postoperative course was uneventful. The tracheotomy tube was removed in ten days. A checkup endoscopic examination performed seven months postoperatively revealed no evidence of stenosis. Both vocal cords moved. The patient has remained free of symptoms to date.

Primary carcinomas of the trachea, though uncommon, may occur anywhere throughout its length. Unfortunately, roentgenograms of the chest show no abnormality in early lesions. Frequently these patients are treated for a tracheobronchitis or asthma until they become inoperable. Any patient with an inspiratory-expiratory wheeze, atypical asthma, or having hemoptysis should be bronchoscoped. Early malignancies of the trachea could be treated similarly to the patient described above by resection and anastomosis. Postmortem dissections show that exposure and mobilization for lesions arising at the level of the manubrium or upward can best be approached through a sternal splitting incision with extension of the incision into the neck. Lesions lower down can best be approached through a right posterolateral thoracotomy incision.

Most carcinomas involving the trachea are secondary extensions from carcinoma of the thyroid, esophagus or bronchi. All such patients should be esophagoscoped at the time of the bronchoscopic examination to determine the status of the esophagus.

As has been noted by others[23] carcinoma of the right upper lobe and main stem bronchus have a tendency to involve the distal trachea and carina early in its development because of the shortness of the right main bronchus and proximity of the upper lobe orifice. Frequently this extension is apparent at the time of bronchoscopic examination but occasionally it may be largely extrabronchial and not apparent until the time of the dissection. Until recently this bronchoscopic finding has been a contraindication to operation.

Until earlier diagnosis of bronchogenic carcinoma can be made the only hope of removing such carcinomas with an adequate margin of safety is by extending the field of operation to include the distal

trachea, carina and portions of the contralateral bronchus if necessary. This more extensive type of resection has now been performed in four instances. All of these patients had bronchogenic carcinomas arising in the upper lobe bronchus. In two of the patients the carcinomas were small but tracheal involvement was apparent on the right lateral wall at the time of the bronchoscopic examination. In the other two the distal trachea was infiltrated as a result of extrabronchial extension.

In cases 1 and 2 the residual elliptical defect in the trachea and left main bronchus following resection was small enough to allow transverse closure after the method of Abbott.[24] In retrospect, it is felt that sufficient mobilization could have been attained in these two patients to have allowed complete resection of the trachea and reimplantation of the distal left main stem bronchus into the divided trachea after the method of Grindlay and Clagett. However, in cases 3 and 4 the residual defect was so long that it was impossible to attain sufficient mobilization of the trachea and left bronchus to allow either complete division and reimplantation or transverse closure of the tracheobronchial opening. Consequently, the defect was closed in a longitudinal direction with individual suture with some resulting narrowing of the left bronchus but not to a degree that any significant stricture developed (In this type of case where part of the tracheobronchial wall can be maintained to relieve tension a better closure without restriction of the bronchial lumen might be attained by using an inlay dermal graft reenforced with wire). Three of these patients are now alive and well. The longest survival being two years. The fourth patient, one who had the most extensive carcinoma, died five months postoperatively.

SUMMARY

A historical review of methods employed in the surgical repair of tracheobronchial defects is presented. Resection followed by primary anastomosis when sufficient mobilization of the trachea is possible appears to be associated with fewer complications than when various types of tubes or grafts are employed to bridge the defect. The surgical management of five patients upon whom operations upon the trachea were performed is presented.

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DISCUSSION

DR. HANDELSMAN (Baltimore): I think Dr. Kay is to be congratulated. The trachea being the last unpaired organ in the body to withstand the assault of the surgeon, it has been a subject of much interest.

The interest of surgeons must, of course, be tempered by the experience of endoscopists, and otolaryngologists who managed, for many years, to keep the patients alive over extended periods of time. Dr. Broyles had an experience where a patient with a proved carcinomatous condition was kept alive for eleven years; he succumbed after eleven years.

Two cases which we have had were of interest recently; one was a patient of Dr. Richard Kieffer: a fifty-six-year-old man who had a lesion in the mid-portion of the trachea. The other was a forty-one-year-old man, who had a lesion involving the lower half of his trachea down to a point within one centimeter of his bifurcation.

In both instances, extensive resection of the trachea was necessary. Repair was undertaken in both instances by the utilization of tantalum mesh overlain by cutis graft. The operative procedure in both cases was quite uneventful, and the postoperative course uneventful.

Unfortunately, we share the experience of many others, in that due to a technical error, the first patient succumbed two weeks postoperatively. The tantalum was not oversewn by the graft, but apparently the edge of the tantalum eroded the right common carotid artery, and the patient succumbed immediately.

The second case is two months postoperative. The man is well and alive. He had four inches removed, including the roof of the right main bronchus replaced by tantalum mesh, and this completely oversewn in all directions by graft.

Certainly, we have been of the same impression that Dr. Kay has, and limited experimental work in dogs has borne out the feeling that what can be accomplished by primary suture is best accomplished that way. The time is markedly lower, and the trauma to the patient is tremendously less. The bifurcation of the trachea having posed the most formidable anatomic problem to surgery, it seems its reconstruction can be carried out by primary suture, approximating the lower bronchial borders, and then—if the defect is too long—we can construct, we feel, tantalum mesh and skin prosthesis, or a number of other ways that have been mentioned by Dr. Kay, and extensively studied by others.

Certainly, Dr. Kay is to be congratulated. I am sure that his series is the largest to date, and we may feel that we are somewhat encouraged to optimism as regards a cure for a disease which must eventually be fatal if radical resection is not carried out.

Society Proceedings

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Meeting of Monday, March 5, 1951

THE PRESIDENT, DR. SHERMAN L. SHAPIRO, IN THE CHAIR

Hemostasis in Otolaryngology Using Gelfoam Powder

MYRON HIPSKIND, M.D.

(Abstract)

Control of bleeding is one of the most challenging problems encountered by the otolaryngologist. Technical difficulties peculiar to this field of surgery and to the treatment of diseases of the ear, nose and throat frequently preclude the use of sutures and ligatures. Hemostasis obtained by the application of pressure and by the use of packs is not commonly practical.

Bleeding from ruptured or traumatized telangiectatic vessels in Kiesselbach's area is controlled easily by coagulation of the point of hemorrhage with a chromic acid bead or live cautery. The success of the procedure is dependent upon direct vision of the area and the application of the cauterizing agent in a dry field. Epistaxis secondary to liver disease associated with either hypoprothrombinemia or vitamin K deficiency, or both, presents a more vexing problem. It is difficult to obtain a dry field in these cases. Bleeding is active and persistent. Should spontaneous arrest occur the exact point of hemorrhage is not easily identified. Recurrence of bleeding is certain. The point of bleeding in these cases is most commonly located in the middle or posterior third of the nose. Direct inspection and treatment is not possible.

During the winter months, the incidence of epistaxis is increased. This is thought to be the result of excessive drying of the nasal mucosa in overheated dry homes and offices. Again, in these cases bleeding is most often from the deeper nasal chamber areas. Similar bleeding is usually seen in hypertensive epistaxis.

Bleeding following the removal of the packing is the primary indication for development of a method that overcomes this unpleasant incident after polypectomy. Bleeding and discomfort on removal of packing from the maxillary antrum leaves much to be desired in postoperative management. Occasionally bleeding will occur after complete and carefully executed adenoidectomy. At this time the surgeon desires a simpler form of hemostasis than the postnasal tampon. The removal of aural polyps, granulation tissue and cholesteatomatous material from the middle ear and surgery of the temporal bone are commonly associated with annoying bleeding. Packing is contraindicated.

The gelfoam "patch" and the insufflation of gelfoam powder have been helpful in the clinical management of all these conditions. The gelatin sponge is prepared from ordinary commercial gelatin by introducing air to obtain the desired porosity. This is heated in an oven until dry. A crisp white material is thus obtained which can be cut into any desired size, or powdered. It is packaged in glass jars, which are subjected to dry heat sterilization at 290° F. before sealing. The gelatin sponge is capable of absorbing 50 times its weight of water or 45 times its weight of blood. It is nonantigenic and nonpyrogenic.

My interest in the application of the method to the field of otolaryngology followed the motion picture demonstration of control of hemorrhage with a gelatin sponge by Dr. H. P. Jenkins at the meeting of the American Medical Association in 1946. The patch technic for the control of epitaxis formed the basis of a report made by Cope and myself the following year.

The difficulties experienced in introduction of the gelfoam patch into the deeper areas of the nasal chamber were multiple. Commonly the pack would be dislodged or become displaced in the process of withdrawing the forceps. Since using the powdered gelfoam we have in part surmounted this problem. We are now engaged in the construction of an insufflator.

It should be emphasized that these absorbable hemostatic agents are not a substitute for ligature or suture in the ordinary routine technics of operative surgery. These new materials are particularly useful when a ligature or a suture is not adequate or feasible.

DISCUSSION

DR. SAMUEL SALINGER: During the past winter we have seen a number of cases of nasal hemorrhage in elderly patients with hypertension, in which repeated packing, left in the nose for as long as

seven days, frequently failed. I wonder if Dr. Hipskind has had any experience with his procedure in this type of case.

The simple bleeding from Kiesselbach's area is not a problem. Our difficulties arise in those cases in which severe bleeding prevents visualizing and locating the bleeding vessel. It is easy enough to say "locate the bleeding point and apply the cautery" but, in actual practice, this is frequently impossible because of the profuse bleeding and the inaccessibility of the vessel.

DR. O. E. VAN ALYEA: Is this advocated for post-tonsillectomy bleeding and as an immediate procedure following operation; does the essayist think the method aids healing?

DR. MYRON HIPSKIND (closing): The gelfoam patch becomes a very pliable and elastic tissue that has been demonstrated to be absorbed in a sterile field in four to six weeks. At the end of the first week it is easily identifiable, but as weeks go by it is absorbed, so that it cannot possibly produce pressure; it becomes part of the tissue. Abundant work has been done by Jenkins and his group to show that if the patch holds for ten minutes to one hour in the abdomen it can be expected to continue to control hemorrhage. Even the hemorrhage in large wounds have been so controlled. As the patch is applied directly to the point of bleeding it and the tissue become, as it were, one structure. However, should a clot form between the site of hemorrhage and the patch, a blowout usually occurs.

As Dr. Salinger stated, bleeding from Kiesselbach's area is easily controlled, but the bleeding we are concerned with is that which continues to recur in spite of packing for a long time. These cases are usually associated with alcoholism, liver disease or hypoprothrombinemia. We have used this technic in a case of persistent bleeding associated with liver disease. The bleeding could be seen but we were unable to control it by the usual methods.

In answer to Dr. Van Alyea, we have tried both the powder and the patch following tonsillectomy. It has been without value. On one occasion the powder was insufflated onto a persistent bleeding adenoid and the bleeding stopped. It is assumed that it was the agent that controlled the bleeding.

Mixed Tumor of the Inferior Turbinates

SAMUEL SALINGER, M.D.

In June, 1949, we reported an unusual tumor of the inferior turbinate (Annals O. R. & L., 58:485 (June) 1949) concerning the classification of which there was some difference of opinion. It

was originally termed an adamantinoma or ameloblastoma and later designated as a mixed tumor in the opinion of pathologists of the Army Institute of Pathology, to whom a slide had been submitted. At that time we stated that although the healing was complete, we feared the possibility of recurrence which, in this type of growth, is not infrequent.

Our fear was not unfounded. About 12 months later the patient reappeared complaining of recurrence of nasal bleeding and, on examination, a spongy friable mass filling the inferior meatus on the previously operated side was found. X-rays failed to show involvement of the bony naso-antral wall or the maxillary sinus. Nevertheless, with the previous history in mind and being aware of the tendency for these growths to recur, we decided on radical exenteration. A Denker operation was performed, removing the entire naso-antral wall including the inferior and middle turbinates, as well as the lining of the maxillary sinus. This was followed by an intensive course of irradiation. Recovery was complete and today (about 18 months) the antrum which is readily visible through anterior rhinoscopy is perfectly smooth and clear.

Histologic study of the excised tissue reveals substantially the same sort of structure as in the original specimens. Masses of basal-like cells are seen arranged at times in columns and groups separated by typical fibrous meshwork. In some areas a more differentiated type of epithelial cell is seen arranged in rows, at right angles to the mass of basal cells.

To understand the genesis and morphology of a tumor of this sort, it is important to bear in mind that the primitive buccal epithelium of the first branchial cleft subsequently differentiates into six different types of epithelium, namely, squamous cells, mucus glands, salivary tissue, enamel organ, thyroid tissue and Rathke's pouch. This has been most clearly explained by Ward and Hendrick in their excellent treatise on "Tumors of the Head and Neck" (p. 305, Williams & Wilkins, 1950). Knowing this, it is not too difficult to explain late development of rests of the primitive epithelium in areas remote from the situs where they normally belong. This also explains the appearance of thyroid and salivary tissue in the nasal and pharyngeal cavities. The reason why it is difficult at times to accurately name a particular growth or give it an exact designation, is the fact that the cells comprising the body of the growth have not differentiated sufficiently to become typical of this, that or the other organ. Unless the morphology approximates that of the enamel organ, the thyroid or the salivary apparatus, one is not justified

in calling the tumor an adamantinoma, a thyroid rest or a salivary gland tumor. As a matter of fact, one would be justified in calling the undifferentiated tumor comprised chiefly of basal cells, as in the case herewith reported, a basiloma, which it simulates very closely. Certainly this case does not exhibit the characteristic histologic picture of adamantinoma, since stellate cells are nowhere to be seen and the stroma fails to show the hyaline substance which is the forerunner of an enamel substance.

On the other hand, the presence of different types of epithelial cells, at times in eccentric arrangements with suggestion of glandular elements here and there, would impel one to call it a mixed tumor, which was the diagnosis given by the pathologists of the Army Museum of Pathology.

However, in the final analysis, it is not the terminology that is so important. What really matters is whether it is benign or malignant. Experience has shown that all these tumors, whether we call them adamantinoma, basiloma, mixed tumor or what not, though primarily benign, are prone to recur locally and infiltrate adjacent tissues, causing extensive destruction and ultimately becoming malignant. In one case, an adamantinoma of the maxilla which was followed for five years postoperatively without evidence of recurrence, turned up after eight years in another city with an extensive growth of the same character in almost the same location. The lesson to be learned, therefore, is that any atypical growth about the oral cavity that exhibits histologically embryonal cells, should be dealt with in a radical manner. The excision should be wide enough to insure complete removal into definitely healthy areas. Experience with mixed tumors of the parotid and adamantinomas of the maxilla amply support this contention.

DISCUSSION

DR. JOSEPH G. SCHOOLMAN: In clinical oncology we do not differentiate too closely between the pathologic terminology of benign and malignant, because we find that pathologically benign lesions are often clinically malignant. For that reason we would like to see the Denker operation placed in the same category as simple mastectomy—a procedure no longer performed. We think the procedure gives too little postoperative view of the area to adequately follow these people, and too often there is recurrence at too advanced a stage before it becomes evident by examination through the small opening allowed by the Denker, to adequately cope with it. We feel that these cases all deserve at least a subtotal resection of the maxilla; the hard palate and the superior alveolar ridge

should be resected so that the entire area is in view. The prostheses now available keep such a procedure from being deforming, or an inhibiting factor in surgical approach. We feel that adequate exposure of the area is the only way to prevent malignant lesions from getting beyond us, and benign lesions or pathologically benign lesions from becoming clinically malignant.

DR. SAMUEL SALINGER (closing): I think Dr. Schoolman's point is well taken. It depends a good deal on the site of the growth. I agree that many times lesions are pathologically not malignant, yet clinically behave as such. In a case such as ours, where the lesion involved only the inferior turbinate, the Denker procedure exposes not only the floor of the nose but the antrum cavity as well. There is no question but that wide exposure is desirable in any case.

We have a case now of a growth on the septum, a fungating bleeding lesion, and are prepared to do a radical operation if the frozen section proves it to be malignant. It goes without saying that the extent of the surgery depends on the type of the lesion as well as its extent.

Meeting of Monday, February 5, 1951

THE PRESIDENT, DR. SHERMAN L. SHAPIRO, IN THE CHAIR

Deafness Due to Mumps: Rehabilitation

ARTHUR J. COOMBS, M.D.

(Abstract)

The author reports a case of deafness in a 36 year old female due to mumps which was bilateral at the onset, but partial recovery occurred on the left side. This side had been previously affected to a moderate degree by a middle ear infection of long standing. In the absence of any previous record of the degree of impairment it is assumed that recovery was complete to the level of the pre-existing loss in this ear. The right ear, which had always been normal, showed practically total loss, which was permanent. There was no vertigo, but the vestibular function in the right ear was greatly diminished.

By means of a hearing aid and a telephone amplifier the patient underwent complete rehabilitation in her position as a private secretary. The hearing aid is suspended high on the right side of her chest so that the telephone may be held conveniently between the

mouth and the microphone of the aid. Photographs show the ease with which this is done. She also studied lip reading as a further step in rehabilitation. It is suggested that many unilateral cases of deafness of unknown etiology in children might be due to mumps.

In the discussion of rehabilitation the following points are made:

1. Subtotal deafness due to mumps is not an insuperable handicap.

2. Psychotherapy should be directed to the patient, if need be, to create a desire to overcome the handicap.

3. Close co-operation between a deafened person and his business associates should be effected whenever possible.

4. Rehabilitation by means of a properly fitted hearing aid, by the study of lip reading and by the use of a telephone amplifier will restore many individuals to their normal fields of activity.

DISCUSSION

DR. FRANCIS L. LEDERER: One is perhaps repaid for his efforts when one hears a physician—no less an otologist—describe what should be considered a very unrewarding part that he has played in the life of a hard of hearing patient. On the other hand, Dr. Coombs has underestimated his role. Undoubtedly he was rewarded by the response of this patient, who had a background of stability, who was able to take a crisis in her life in her stride. We should not forget that a patient who is deprived of a special sense reacts as he or she would when deprived of anything in earliest childhood. Perhaps Dr. Coombs was favored in having a patient with a good background. However, the employer, too, realized her importance as an employee, and he has discovered the rewards of medical therapy (audiology) in dealing with deafness, in which the otologist is playing a more and more important part in the rehabilitation of the deaf and hard of hearng patients.

Dr. Lindsay reported 16 cases of unilateral deafness and, when one thinks of statistics, that is rather a small incidence in mumps deafness that Dr. Coombs has reported from the literature. Many people go through life with unilateral deafness without realizing it until something brings it to their notice. So I believe that the statistics are not completely valid. One would say that many cases of so-called unilateral deafness may be mumps encephalitis, because so many people who have had mumps are not aware of it. The role of rehabilitation in bilateral hearing loss is, as Dr. Coombs has emphasized, vital to the patient.

DR. ALFRED LEWY: One of my patients, an ex-nurse, has discovered that she can use her hearing aid by laying the microphone on the telephone; this avoids the rubbing noises. Several other patients have complained that they could not use it through their clothing. They still have rubbing noises because their hands are not perfectly still. A number of these patients are as deaf as those reported by Dr. Coombs, and perhaps he has some suggestions to offer.

DR. ROBERT HENNER: I would like to commend Dr. Coombs on this splendid paper. He called attention to the fact that there are two phases of mumps when ear symptoms are present. In my experience the early symptoms are usually catarrhal otitis in origin; in the second phase, are associated with nerve deafness and labyrinthitis. I have seen only one case which had deafness and labyrinthitis. This patient had spontaneous nystagmus at the height of the disease. This made it improbable that the cause of deafness would be overlooked.

DR. JOHN R. LINDSAY: At one of our national meetings a few years ago the statement was made by the head of a large infectious disease institution, to the effect that he had never known deafness to result from mumps. That experience we know could only be explained on the basis that an adequate examination for unilateral deafness had not been made. Among the employees of our own institution we have seen three cases of unilateral deafness develop in a period of a few years from mumps. About three years ago I observed two students in the same class in college with unilateral deafness from mumps. I would agree with Dr. Lederer that it is a common complication. In my experience very few of these cases have had more than a day or two of dizziness at the onset. All have had very good function of the vestibular mechanism in the affected ear. In the older textbooks there are cases described with vestibular function diminished or lost, but I have not seen this. They have all maintained good vetibular function, but had profound deafness for air and bone conduction.

DR. ARTHUR J. COOMBS (closing): I wish to thank each of the discussors for their kind words and helpful suggestions. Dr. Lederer emphasized the point of teamwork between this young lady's employer and herself. I think the doctor can play an important role here. Dr. George Coleman happened to know the employer and brought a lot of influence to bear in getting his co-operation, and giving the patient all the time necessary to become adjusted.

She was not bothered by rubbing noises. There was no vertigo, but she had definite diminution of vestibular function. As Dr. Lindsay emphasized, unilateral cases of deafness in undetected mumps is more common than we think.

I found nothing new which would help regarding microbiology. It was always termed a virus type of infection.

Bronchoscopic Aspects of Fibrocystic Disease of the Pancreas

PAUL H. HOLINGER, M.D.

GEORGE C. ANISON, M.D.

AND

KENNETH C. JOHNSTON, M.D.

(Abstract)

Fibrocystic disease of the pancreas has been recognized as a clinical entity for only the past 11 years. It is of great importance to the bronchologist because the associated broncho-pulmonary suppuration is usually the cause of death. The condition is a congenital anomaly of the pancreas and other glandular structures, such as the bronchial and intestinal mucus glands, that results in an absence of fat digestion and absorption and, consequently, an avitaminosis of the fat soluble vitamins.

Fibrocystic disease of the pancreas is characterized by failure of the infant to gain weight, general debility, streatarrhea with large, foul stools, and an extensive bronchopulmonary suppuration. Diagnosis is established by absence of trypsin in the stools, the flat vitamin A curve in the blood, and the absence of pancreatic enzymes in the duodenal secretions of infants who show the clinical picture described above of marked debility, streatarrhea and absence of subcutaneous fat.

During the past ten years, 74 infants who have had fibrocystic disease of the pancreas have been seen at the Research and Educational Hospital and the Children's Memorial Hospital. Of these, 32 required bronchoscopic aspiration to relieve them of their obstructing secretions; many had been admitted originally under the diagnosis of bronchopneumonia, atelectasis or bronchiectasis, and the final diagnosis was established only after admission and study.

The tracheobronchial tree of these infants showed a severely inflamed tracheal and bronchial mucosa with the lumina filled with thick viscid mucopus. Bacteriologic examination of this material usually showed a pure growth of staphylococci, but other organisms were frequently found as well.

Therapy from a bronchoscopic standpoint consisted of efforts to promote endobronchial drainage through steam inhalations, postural drainage, and bronchoscopic aspiration. Chemotherapy was administered by aerosol and injection. Aureomycin orally was found particularly beneficial. Dietary management consists of the administration of approximately twice the usual caloric requirements of the patient's age and weight. A high protein, low fat and low carbohydrate diet has been suggested, but a regular, unrestricted diet is now generally given. Certainly high doses of vitamin A, C, and D are indicated, together with liver extract. Pediatricians suggest that pancreatic granules be added to the diet, but since it decreases the appetite this procedure has been questioned. Recently cortisone has been administered because of its action as a carbohydrate metabolizer.

The prognosis in cases of fibrocystic disease of the pancreas is generally not favorable. The frequent recurrences of the bronchial infection are generally responsible for terminal bronchopneumonia. However, with active treatment of the bronchopulmonary suppuration many infants improve, and therefore repeated bronchoscopic aspiration is an important factor in prolonging the lives of these infants.

DISCUSSION

DR. PAUL H. HOLINGER: The etiology of this condition is completely unknown. All measures so far are completely palliative and actually have no effect on the course of the disease. The pulmonary disease runs concurrently with the pancreatic disease and is not a part of the pancreatic disease.

The prognosis is usually poor. Some children have lived for five or six years, and a few eight to ten years. Usually such children have been treated shortly after the disease was recognized. While the actual etiology is unknown, there is a familial tendency with infants dying one after another, to the point where the mother is advised to have no more children. This tendency is definitely recognized, and the family should therefore be warned.

Planes of Surgical Dissection of the Neck

MAURICE F. SNITMAN, M.D.

AND

BURTON J. SOBOROFF, M.D.

(Abstract)

Surgical dissection of the neck must necessarily be based on an intimate knowledge of anatomy. There are so many varied descriptions of the structure of the neck that it is sometimes difficult to interpret them. This is particularly true with regard to the fascial layers.

The cervical fascia consists of two chief layers, superficial and deep. The superficial fascia consists of fatty tissue and includes the platysma muscle within it. The deep cervical fascia is made up of an outer layer or superficial layer; a middle or omohyoid layer; and a deep layer. Descriptions of the outer fascial layer, particularly in relation to the parotid, are especially variable; those of the middle layer of the deep fascia are even more so. The deep layer of deep cervical fascia is generally described as the prevertebral layer. It lies immediately over the bodies of the cervical vertebrae and prevertebral muscles and extends laterally to the transverse processes of the cervical vertebrae.

Weintraub, in 1941, presented a description of a "new anatomic and functional systematization of the connective tissues of the neck." He considered the connective tissues in three major classes rather than individual, minutely subdivided fascial sections. These included:

1. Nerve-vessel supporting connective tissue
   a. Nerve-vessel bundles about main trunks.
   b. Nerve-vessel sheets about branches.

2. Periorgan connective tissue. This included the condensation of the loose areolar tissue about individual organs in the neck.

3. Areolar tissue spaces, made up of loose areolar "packing" tissue filling the spaces between the muscles, vessels and organs in the neck.

Dissection of the neck in the fresh cadaver and in the living patient reveals this to be the arrangement of the connective tissues of the neck.

These points were demonstrated with slides, and kodachrome photographs of anatomic dissections.

DISCUSSION

DR. MAURICE F. SNITMAN: An excellent example of a misconception of fascial structure is provided by the so-called fascial capsule of the submaxillary salivary gland. After elevation and retraction of the skin and platysma flap over the submaxillary triangle, the loose tissue thus exposed is placed under tension because of elevation of the mandible. This collapsed and stretched loose areolar tissue gives the appearance of a distinct fascial layer. In reality this is an artificial structure, as its true loose areolar structure can be demonstrated by relaxation of this fascia and teasing it with tissue forceps.

This anatomic interpretation holds true for most of the hitherto accepted fascial layers of the neck.

Tracheotomy in Barbiturate Poisoning

ROBERT B. LEWY, M.D.

AND

J. W. SIBBITT, M.D.

(Abstract)

The basic therapy of patients suffering from acute barbiturate poisoning is essentially the responsibility of the internist. It is, however, essential that the otolaryngologist and anesthetist play a part in maintaining an adequate airway in these deeply comatose individuals. The physiopathology which occurs in barbiturate poisoning is as follows:

1. There is a basic physiology of respiratory obstruction regardless of the type. In any simple obstruction of the trachea there first occurs reduction in ventilation capacity of the lungs. This decrease involves hypoventilation which, as it becomes more severe, results in specific chemical changes in the blood:

   a. Retention of carbon dioxide (hypercapnia)
   b. Fall in oxygen saturation of arterial blood (anoxemia)
   c. The above two produce fall in the blood pH (acidemia)

These three factors constitute the phenomenon asphyxia. In early obstruction there is a strong respiratory stimulus. In longer

obstruction, a critical point is reached whereby these blood changes cease to act as a stimulus and the breathing effort weakens. This causes an increased asphyxia which, in turn, actually inhibits breathing, thus setting up a vicious and progressive cycle. Death in these cases is usually due to respiratory failure.

Treatment is directed toward a maintenance of adequate respiratory exchange and blood pressure levels by means of oxygen, analeptics, convulsants such as picrotoxin and metrazol, and intravenous fluids or plasma with ephedrine. The laryngologist and anesthesiologist should be called upon to aid in maintaining oxygenation and an adequate airway. Simple suction in the hypopharynx plus postural drainage may be adequate to remove secretions which pool in the hypopharynx and the tracheobronchial tree. If depression is prolonged further procedures may be required. Bronchoscopy may be necessary; an endotracheal tube may be inserted. If cough and gag reflexes have not returned in eight to 12 hours, tracheotomy should be done. If the edotracheal tube is allowed to remain in place for a prolonged period of time serious damage to the larynx may occur.

Three illustrative cases are presented in detail, in two of which treatment was successful. In Case I, two errors were noted: the laryngologists were not called upon until trouble had arisen with the rubber airway tube; trauma to the larynx resulted. In Case II, the same two problems were present. In both cases earlier tracheotomy would have been the procedure of choice. In Case III, based upon knowledge gained from the first two cases, the laryngologist was called immediately. The tube was removed before it could do any harm, yet it served a necessary and vital purpose. Tracheotomy was not required due to improvement of the patient. Death was attributed to barbiturate poisoning with secondary pneumonia.

CONCLUSIONS

1. A trained team of internist, anesthesiologist and laryngologist is required for prompt, proper, and adequate treatment of barbiturate poisoning, all of whom together should see the case as an immediate emergency.

2. An immediate estimate of airway adequacy should be made and, if obstructed, this must be cleared immediately.

3. If cough and swallowing reflexes are absent, and if the patient is unconscious and a long period of unconsciousness is anticipated, immediate tracheotomy is indicated.

4. If it appears that the patient may improve to a reflex level in eight to ten hours, temporary intubation with an anesthesia tube, oxygen and maintenance of airway is desirable, with re-evaluation later by the medical team.

DISCUSSION

DR. HANS VON LEDEN: I would like to congratulate Dr. Lewy on his lucid description of the problem, and to stress early tracheotomy, because I feel as he does that a stormy course of two or three days may often be avoided.

I wish to show a slide on another patient. This patient was brought to the emergency room with a diagnosis of carbon monoxide poisoning. He had been found in his garage at 10:00 A. M. with the motor of the car running. Artificial respiration was instituted and the usual supportive measures were given. That evening it was learned that a bottle of barbiturates was missing and that the patient had been in the garage only a short time before he was discovered. He became progressively worse; by 10:00 P. M. the temperature was 106 and the pulse and respiration 160 and 62 respectively; he was in complete coma. The chest and throat were full of secretions which were aspirated almost constantly through an endotracheal tube, but there was a rapid flow of secretion, possibly due to irritation by the carbon monoxide gas. A tracheotomy was performed about 11:00 P. M. and the supportive measures were continued. It was then possible to aspirate secretions promptly and effectively and to supply the needed oxygen. The remainder of the course was entirely uneventful.

I would like to thank Dr. Lewy again for bringing out these important points, and stress that early tracheotomy often means the difference between an easy and a stormy course.

DR. JOHN ELSEN: In addition to the advantage of a tracheotomy mentioned by Dr. Lewy, the evaluation of the cough reflex is very easily made after tracheotomy. The cough reflex on stimulation of the tracheal mucosa is one of the last reflexes to disappear in severe barbiturate poisoning. The presence of the cough reflex is extremely important, whether or not tracheotomy is done. If the cough reflex is absent or inadequate, secretion will collect in the smaller branch bronchi, and never be brought to the trachea or main bronchi where it can be aspirated.

With tracheotomy, the aspirating catheter can be inserted beyond the cannula and the activity of the cough reflex can be estimated easily. If there is little or no cough, there is danger of atelec-

tasis and the patient may drown in his own secretions. The dosage of picrotoxin can then be altered accordingly.

Dr. Paul H. Holinger: Certainly the increasing use of tracheotomy for almost everything warrants comment. Dr. Lewy has added an indication to the many disease processes for which it is being used. Again we have to stress to our medical colleagues that tracheotomy is a safety valve. In the last two years we have brought this out with one condition after another—polio, postoperative collapse, coma, true cerebral accidents, etc., and it again brings out the function of the anesthesiologist taking over the management of these conditions to the elimination of the otolaryngologist, in a field in which we think we have more or less priority and in which we feel we should be able to give the necessary treatment.

The use of an indwelling tube is non-surgical procedure; frequently it is desirable to use this to avoid surgical procedures, but Dr. Lewy has shown the damage that can occur. We have seen many times various complications from indwelling tracheal tubes. If the anesthesiologist inserts a tube, from then on the otologist should take over. Certainly, 12 hours is the maximum time the tube should be permitted to stay in, and after that time a tracheotomy should be performed.

I think Dr. Lewy's cases are extremely interesting. We might point out that in the third case, where the otolaryngologist was called early, the patient was lost. I wonder why, after bronchoscopy, the tube was inserted and aspiration carried out.

We have become used to using the silver tracheotomy tube almost to the exclusion of other means. However, small tracheal tubes which can be attached to a bag for respiration are much superior. The respirator, if necessary, can be handled more easily than with the silver tracheotomy tube that is used routinely.

Dr. Robert B. Lewy (closing): I am much pleased to have had this excellent discussion, which throws additional light on the subject. Both Dr. Sibbitt and I, who did part of the tracheotomies, were well aware of the fact that the third patient did not survive in spite of the fact that he was seen early by a laryngologist. I think this patient died independently of any airway obstruction as such. The question of clinical judgment must come into every case. I would defend not doing tracheotomy on this patient; in view of his condition and the presence of reflexes it was not indicated. His death was not due to drowning or obstruction. It was for that

reason that this case was chosen to present; we were anxious to show that cases do not invariably require tracheotomy and we think it would be a mistake to give that impression.

I could not agree with Dr. Von Leden more. He has simply emphasized the necessity in another condition for doing tracheotomy. We generally believe that picrotoxin is best, but even that can lead one astray.

Abstracts of Current Articles

EAR

Studies in Auditory Fatigue and Adaptation.

Hood, J. D.: Acta Oto-Laryngologica, Supplementum 92, London, 1950.

The author recognizes two different types of fatigue: post-stimulatory which occurs following stimulation and per-stimulatory or adaptation which occurs during auditory stimulation. The latter has received little attention. A self recording and self operating audiometer of the Bekesy type is used to measure fatigue. After testing three normal subjects Hood concludes that between 90 and 95 db. there exists a critical fatiguing intensity below which fatigue is slight and above which it increases rapidly. He further believes that the pain threshold is not a safe guide to the onset of serious pathological changes in the cochlea. His results indicate that there is no critical duration for a fatiguing tone; it is rather the intensity than the duration which determines the difference between auditory fatigue and stimulation deafness, the former being physiological and the latter pathological.

Hood substantiates experimental findings of Derbyshire and Davis on equilibration by showing that maximum fatigue occurs when stimuli frequencies of 900, 1800 and 2700 are used. Fatigue occurs in the end-organ, and a damaged inner ear is more susceptible to fatigue than a normal ear, according to results obtained from three patients. The duplicity theory of hearing is supported by two experimental findings: a. that the lower frequency limit of fatigue remains constant with intensity while the upper limit becomes progressively higher; that the frequency distribution of fatigue varies with intensity and extends above the stimulus frequency with high intensities.

Using loudness balance testing the author determines per-stimulatory fatigue not only of the threshold receptors but other nerve elements acting at higher intensity levels. This type of fatigue reaches its maximum in about three and a half minutes and is inde-

pendent of the intensity of the stimulus. Its recovery is most rapid in the first ten seconds and is complete after one minute. The total fatigue in any receptor group of specific size is independent of the intensity of the stimulus. Hood investigated this type of fatigue in twenty five patients with unilateral Ménière's disease.

SATALOFF.

NOSE

The Management of Acute Frontal Sinusitis.

Goodale, R. L.: New England Journal of Med. 244-23 (June 7) 1951, pp. 877.

This excellent article reviews the present trends in the treatment of acute frontal sinusitis, paying particular attention to the impact of antibiotic therapy on this condition. He states that the fundamental picture has not been altered, that only new and more effective measures are available. He emphasizes the danger inherent in the masking of symptoms and rightly stresses the importance of definitive bacteriology and especially selectivity cultures. One should never place entire reliance on this form of therapy. Undrained pus still requires evacuation and serious complications may ensue if this be neglected. Simple trephine usually is the procedure of choice although extension of the process may indicate more radical measures. Indiscriminate use of antihistamines has little or nothing to offer, unless there is a definite allergic factor.

HILL.

The Use of Antibiotics and Vasodilators in Atrophic Rhinitis (Ozena).

Sternstein, H. J.: N. E. Journ. Med. 245-5, pp. 165 (Aug. 2) 1951.

The author reports a new method for the treatment of atrophic rhinitis by combining the local use of antibiotics with vasodilators, given orally and parenterally. 13 cases were studied, for from one and one-half months to one year. The antibiotics employed were streptomycin, penicillin, bacitracin, and gantrisin, depending upon the predominant flora and the response to therapy. Histamine, Priscoline and niacin were used as vasodilators. All nasal irrigations were discontinued and lamb's wool nasal plugs were prescribed to be

inserted three or four times hourly during the day to stimulate secretion.

Four of the cases showed striking improvement. Six were significantly improved and three slightly improved. It was felt that the use of the vasodilator drugs was more effective when used in combination than singly. The increase of the erectile tissue of the inferior turbinates suggested that the changes in atrophic rhinitis may not be entirely irreversible.

Five patients in the group had had previous sinus surgery. These showed a much slower response to treatment and would indicate the undesirability of operation in atrophic rhinitis.

While this is obviously a preliminary report the author feels that this combination of antibiotics and vasodilators can eliminate odor and crusts and "promote a reversal of the regressive changes." How lasting such improvement might be and whether or not treatment must be maintained can only be surmised.

HILL.

THROAT

Closure of Large Laryngotracheal Fistulae.

Rethi, Aurele: *Les Annales D'Oto-Laryngologie* 67:682-687 (Oct.) 1950.

In closing a laryngotracheal fistula, in cases where the fistula has been kept open for a long time, it is necessary to dissect free and elevate the skin within the laryngotracheal stoma. This skin is then turned inward and closed, to help reline the airway. Then one dissects two flaps of skin and subcutaneous tissue, one on each side of the laryngotracheal fistula. Each flap is triangular in shape. One flap has its apex at the upper end and its broad base of attachment at the lower end; the other flap has its apex at the lower end and its broad base of attachment above. Now the closure is completed by transposing the one flap into the bed of the other, anchoring the apex of the one into the lateral extremity of the base of the other. The result is a "Z"-shaped scar, in which the line of closure crosses the midline diagonally. The objective is to avoid the unsightly and constricting vertical midline scar on the anterior aspect of the neck, and to attain a smooth interior surface without constricting the lumen of the airway.

The author then proceeds to describe a second method of closure for a laryngotracheal fistula. In this method one dissects a cutaneous bridge on each side of the stoma. These bridges of skin are turned inward to cover the fistula and present their epidermal surfaces toward the lumen of the airway. The outer raw surface is then covered with a flap of skin, brought up from the anterior chest wall.

HARKINS.

Bronchoscopy in Cardiac Diseases.

Paley, M. P. Y.: Les Annales D'Oto-Laryngologie 67:384-394 (May-June) 1950.

The author has performed over 150 bronchoscopic examinations in cardiac cases in a period of one year. In only five cases, all of these of mitral stenosis with pulmonary edema, was there any complication to the bronchoscopy: in these there occurred dyspnoea, with copious frothy blood-tinged secretion escaping from all the bronchial orifices, as soon as the bronchoscope was introduced into the trachea. These secretions welled up into the bronchoscope and made the examination impossible. Among the later cases in this series, the author did not examine such cases; also he did not bronchoscope patients with aortic aneurysm. He feels that, excepting for these cases, any cardiac patient can have a bronchoscopic examination, if the procedure is done gently.

It is the mucosal changes in the tracheo-bronchial tree which interests the author. He found the tracheal mucosa to be almost always normal in the cardiopathies. On the other hand the mucosa of the bronchi is almost always altered which he attributes to the difference in blood supply. When the same patient has been bronchoscoped more than once, the difference in appearance of the mucosa under rest and medical treatment, has been quite apparent. He concludes that the bronchial mucosa is a very sensitive indicator of the state of the pulmonary circulation. There were cases in which these changes have been the first sign of pulmonary involvement in cardiac cases—cases not yet manifesting dyspnoea, expectoration or any other pulmonary symptoms.

He noted the following changes in the bronchial mucosa: vascular engorgement, edema, hypersecretion. In about half the cases showing vascular engorgement there was bleeding at the slightest

contact of the instrument or sponge. In six of these cases telangiectases were seen. The mucosal edema was in some cases extreme, occupying as much as ¾ of the calibre of the bronchial lumen; the edema was symmetrical and bilateral. Hypersecretion was of two types: globules of dense mucous, sometimes a little purulent, and thin, frothy secretion.

Of the 43 cases of mitral stenosis in this series, 36 presented intense vascular engorgement of the bronchial mucosa. Of these 36, there were 23 with bleeding during the bronchoscopy; 16 of these patients had had hemoptyses previously. Telangiectases were noted in six cases. The author believes that in these cardiac cases, with increased pressure in the left auricle and the pulmonary veins, the anastomoses between the pulmonary veins and the bronchial veins becomes highly developed, and becomes an important route for the blood to flow from the pulmonary veins to the bronchial veins and back into the systemic circuit (superior vena cava). The author believes that this explains hemoptysis in these cases.

HARKINS.

Books Received

Die Nervenkrankheiten.

By Professor Dr. Georges Schaltenbrand, Director of the Neurological Clinic, University of Würzburg. Pp. xx+880, with 538 illustrations and 68 pages of references. Stuttgart, Georg Thieme Publisher, 1951. (Price DM 87.-)

Color Atlas of Morphologic Hematology.

By Geneva A. Daland, B.S., Chief Laboratory Assistant in Hematology, Thorndike Memorial Laboratory; Research Laboratory Technician, Boston City Hospital. Pp. 70, with 23 illustrations, 14 in color. Cambridge, Massachusetts, The Harvard University Press, 1951. (Price $5.00)

Surgical Care—A Practical Physiologic Guide.

By Robert Elman, M.D., F.A.C.S., Professor of Clinical Surgery, Washington University School of Medicine; Assistant Surgeon, Barnes Hospital; Associate Surgeon, St. Louis Children's Hospital; Director of Surgical Service, H. G. Phillips Hospital, St. Louis, Missouri. Pp xviii+586 with 18 figures and 18 tables. New York, Appleton-Century-Crofts, Inc., 1951.

Notices

AAAS PHILADELPHIA MEETING

The 118th Meeting of the American Association for the Advancement of Science will be held in Philadelphia December 26-31, 1951. It will include programs of all 18 of the Association's sections and about 45 participating societies. Focus of the approximately 225 sessions will be Convention Hall, adjacent to the University of Pennsylvania's School of Medicine and its associated hospitals; there will be some meetings in the downtown hotels, especially the Bellevue-Stratford (headquarters) and the Benjamin Franklin (zoologists). There will be numerous features of particular interest to those in medicine and experimental biology.

UNIVERSITY OF ILLINOIS

For the period of the National Emergency, the University of Illinois College of Medicine will offer a combined three year residency training program in Otolaryngology, which will include the basic course material, in fulfillment of Board requirements.

Residents will rotate through the Research and Educational Hospitals, the Illinois Eye and Ear Infirmary, the Hines Veterans Administration Hospital and the various affiliated institutions. The residency will be so flexible that should it be interrupted because of military service, the period of training may be resumed upon returning to civilian life.

Under this arrangement, no course fee is to be involved and in the case of most of the aforementioned institutions, a stipend is provided.

For further information, kindly address; Head of the Department of Otolaryngology, University of Illinois College of Medicine, 1853 West Polk Street, Chicago 12, Illinois.

THIRD PAN-AMERICAN CONGRESS

The Pan American Association of Oto-Rhino-Laryngology and Broncho-Esophagology will hold its Third Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology in Havana, Cuba, in 1952.

For information address: Dr. Chevalier L. Jackson, 3401 N. Broad Street, Philadelphia 40, Pa.

ANNALS

The management of the ANNALS desires to buy, at $1.50 each, copies of the following numbers which are out of print:

March, June, 1940	March, Dec., 1946
March, June, 1943	June, Sept., 1947
March, Sept., Dec., 1944	March, Dec., 1948
March, June, Sept., Dec., 1945	March, 1950

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE AND REHABILITATION OF THE AMERICAN MEDICAL ASSOCIATION

(As of September 1, 1951)

Audicon Models 400 and 415
Audivox Model Super 67
Aurex Model F
Aurex Model H

Beltone Harmony Mono-Pac
Beltone Symphonette Model
Beltone Mono-Pac Model M

Cleartone Model 500
Cleartone Regency Model

Dahlberg Model D-1
Dysonic Model 1

Electroear Model C

Gem Model V-35
Gem Model V-60

Maico Atomeer
Maico UE Atomeer
Maico Quiet Ear Models G & H
Maico Model J
Mears (Crystal and Magnetic) Aurophone Model 200
1947-Mears Aurophone Model 98
Micronic (Magnetic Receiver) Model 101
Micronic Model 303
Micronic Star Model
Microtone Audiomatic T-3
Microtone Audiomatic T-5
Microtone Classic Model T-9
Microtone Model T-10
Microtone Model 45

National Cub Model (C)
National Standard Model (T)
National Star Model (S)
National Ultrathin Model 504
National Vanity Model 506

Otarion Model E-1S
Otarian Model E-2
Otarion Model E-4
Otarion Models F-1, F-2 and F-3
Otarion Model G-1 (Whisperwate)

Paravox Model J (Tiny-Myte)
Paravox Models VH and VL (Standard)
Paravox Model XT ('Xtra-Thin)
Paravox Model XTS ('Xtra-Thin)
Paravox Model Y (YM, YC and YC-7) (Veri-Small)

Radioear Permo-Magnetic (Multipower)
Radioear Permo-Magnetic (Uniphone)
Radioear All-Magnetic Model 55
Radioear Model 62 Starlet
Radioear Model 72
Rochester Model R-1
Rochester Model R-2

Silver Micronic (Magnetic and Crystal) Models 202M and 202C (See Micronic)
Silvertone Model 103BM
Silvertone Model J-92
Silvertone Model M-35
Silvertone Model P-15
Solo-Pak Model 99
Sonotone Model 700
Sonotone Model 900
Sonotone Models 910 and 920
Sonotone Model 925
Sonotone Model 940
Super-Fonic Hearing Aid

Televox Model E
Telex Model 22
Telex Model 97
Telex Model 99
Telex Model 200
Telex Model 300-B
Telex Model 400
Telex Model 1700
Tonamic Model 50
Tonemaster Model Royal
Trimm Vacuum Tube Model 300

Unex Model A
Unex Midget Model 95
Unex Midget Model 110

Vacolite Model J

Western Electric Models 65 and 66

Zenith Model 75
Zenith Miniature 75
Zenith Model Royal

(All the accepted hearing devices have vacuum tubes.)

Accepted hearing aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS

Aurex (Semi-Portable)
Precision Table Hearing Aid
Sonotone Professional Table Set Model 50

OFFICERS

OF THE

NATIONAL OTOLARYNGOLOGICAL SOCIETIES

American Academy of Ophthalmology and Otolaryngology

President: Dr. Derrick Vail, 700 N. Michigan Blvd., Chicago 11, Ill.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Meeting: Oct. 14-19, Chicago.

American Board of Otolaryngology

President: Dr. LeRoy A. Schall, 243 Charles St., Boston 14, Mass.
Secretary: Dr. Dean M. Lierle, University Hospital, Iowa City, Iowa.

American Broncho-Esophagological Association

President: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia, Pa.
Secretary: Dr. Edwin N. Broyles, 1100 N. Charles St., Baltimore 1, Md.

American Laryngological Association

President: H. Marshall Taylor, M.D., Jacksonville, Florida.
Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia 2, Pa.
Meeting: Toronto, Ontario, Canada, May 20 and 21, 1952.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Robert C. Martin, 384 Post St., San Francisco 8, Calif.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.

American Medical Association, Section on Laryngology, Otology and Rhinology

Chairman: Dr. James M. Robb, 641 David Whitney Bldg., Detroit, Mich.
Secretary: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis, Tenn.

American Otological Society

President: Dr. Gordon D. Hoople, 1100 E. Genesee St., Syracuse, N. Y.
Secretary: Dr. John R. Lindsay, 950 E. 59th St., Chicago, Ill.

III Pan-American Congress of Otorhinolaryngology and Broncho-Esophagology

President: Dr. José Gros.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa.